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THE CLASSIFICATION AND CERTAIN PATHOLOGICAL ASPECTS OF CHRONIC ARTHRITIS*

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ONE of the objects of inquiry in medicine is the accurate classification of disease. This may be the first step in developing a new conception or in increasing our knowledge of its processes. There is, perhaps, no branch of medicine in which names have been used with less satisfaction than in the designation of diseases of the joints. There are scarcely two physicians who use the same terminology for arthritis, and when you add to this the varying use of terms by writers in England, other European countries and America, the confusion is obvious. If, however, the history of the names that have been given to the arthropathies is studied, it will be plain that the introduction of each corresponded to some special conception. Ignoring this fact has led to the excitation of many controversies. We find, for example, one malady disguised under many different names, or one term may be used obscurely, meaning sometimes one disease, and sometimes another, or indeed at times a group of separable disorders.

The use of terminology in discussing arthritis will naturally vary with the special interests of the physician. The terms that are suitable for the pathologist may not be satisfactory to the clinician or the investigator interested in etiology. In attempting to clarify this unsatisfactory state of affairs, the American Committee for the Control of Rheumatism has proposed a classification based on the predominating pathologic changes in the joints which were defined some years ago by Nichols and Richardson. The British Ministry of Health and the International League for Control of Rheumatism have likewise adopted similar classifications but the names they use are different and confusion continues. For example, the term Atrophic, as adopted by the American Committee, is used synonymously with Rheumatoid, Primary Progressive Arthritis or Proliferative Arthritis, and the term Hypertrophic Arthritis is used in place of Degenerative, Osteoarthritis, or Arthritis Deformans.

It is necessary, therefore, in the use of any classification to define precisely what is meant by the terms used. From the position of the clinician the use of the classification adopted by the American Committee for the Control of Rheumatism has as its chief attraction simplicity. It seems further agreed that the two broad types proposed include only cases in which a specific cause cannot be proved. From the standpoint of an anatomic diagnosis this classification is satisfactory, but one must not overlook the fact that the diagnosis of atrophic or hypertrophic arthritis should never be accepted as the sole diagnosis until all of the known etiologic factors causing arthritis have been excluded. We must not forget that in Nichols and Richardson's monograph the point was made repeatedly that the two pathologic types described by them (Proliferative-Degenerative) were probably caused by a variety of agents, and the classification proposed was an anatomical one. Thus, when the protocols of their cases are examined we find unquestioned cases of gonococcal arthritis included with proliferative arthritis and a number of Charcot joints in the group designated as degenerative arthritis. I emphasize this to point out the fact that the discrimination of cases on a basis of anatomic findings alone is unsatisfactory and should not be accepted as final until all the diagnostic possibilities have been properly excluded. This is offered in an attempt to excite further interest in increasing the accuracy of our methods of diagnosis. It is not a simple matter to make a precise diagnosis in every case of arthritis even when the etiology can be detected with certainty. If anyone is skeptical of this statement let him read Allison and Ghormley's recent book. They point out that in 41 cases in which the diagnosis of tuberculosis of the joints was made, only 27 or 66 per cent were proved to be due to tuberculosis, and in 42 cases in which it was not considered it was proved to be present in 55 per cent. Their experience is a general one.

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In the face of these facts, and in view of what has been said regarding the confusion of terms I should like to stress the importance of attempting to make an etiologic as well as an anatomic diagnosis. When I refer to etiology it is to be understood that I do not refer to infectious agents alone, but to other conditions such as trauma, static and mechanical defects, errors in metabolism such as in gout, bleeding such as occurs in hemophilia, and other recognizable factors.

It has seemed to me that the study of arthritis has been hampered by a lack of detailed knowledge regarding the anatomical variations in the joint structures with advancing age. For example, are the joints of individuals 35 years of age of the same anatomic structure as those of 65 years? The answer to this question is of importance from the viewpoint of the ultimate changes that may occur as the result of damage from an invasion of a foreign substance; and it also may teach us something about the early effects of injury to joints and provide us with information of value in preventing certain conditions. It is unnecessary to recall the fact that the early stage of any form of joint disease is often not to be detected clinically but if knowing something of the development of lesions in joints we are able to detect them earlier than at present, some advance may be made. Finally, such information is important in order to establish a normal base line.

For these important reasons my colleagues, Drs. Parker, Myers, and Irwin, and I have made a systematic study of 100 consecutive knee joints of patients coming to necropsy, in an attempt to define the changes that might be anticipated at various age periods. Whenever possible, the entire knee joint was removed together with the lower end of the femur and upper end of the tibia. When this was not possible the entire articular surface was removed by means of a saw. A gross description was made and any areas appearing abnormal were studied histologically. At this time I shall confine my remarks to the gross pathologic changes.

There were 67 joints from male patients and 33 from females. Inasmuch as the changes were precisely the same both qualitatively and quantitatively the two groups are considered together.

ANATOMICAL CONSIDERATIONS

It is not my purpose to comment in detail upon the anatomy of the knee joint. Some of the facts it is necessary to know before one can clearly understand the changes which were found. The relevant facts are recalled herewith.

The articulations of the knee joint are divided into groups: (1) the articulation of the patella with the femur; (2) the articulations of the two condyles of the femur with the corresponding meniscus and condyles of the tibia.

The articulation of the patella with the femur is partly a gliding one, but inasmuch as the articular surfaces are not mutually adapted to each other, the movement is not a simple gliding one. An appreciation of this fact will become obvious when the various movements of the knee joint are analyzed in detail.

For purposes of describing the movements of the patella, and its articular surface, it may be divided indistinctly into seven facets, the upper, middle and lower horizontal pairs and a median perpendicular facet.

The surface of the femur which articulates with the patella is named the patellar or anterior surface. It presents a median groove which extends downward to the intercondyloid fossa and two convexities, the lateral of which is broader, more prominent and extends farther upward than the medial. In the extended position of the leg, when the quadriceps femoris is relaxed the patella lies loosely on the front of the lower end of the femur. When the knee is forcibly flexed, the median perpendicular facet is in contact with the semilunar surface of the lateral part of the medial condyle of the femur; this semilunar surface is a prolongation backward of the medial part of the patellar surface of the femur. As the leg is carried from the flexed to the extended position, first the upper pair, then the middle, and finally the lowest pair of horizontal facets is successively brought into contact with the patellar surface of the femur. In the process of walking, in which the leg is alternately extended and semiflexed, the part of the patella which is brought into contact with the femur is the median pair of horizontal facets. The importance of this will be stressed presently.

The lower end and posterior parts of the articular surface of the femur constitute the tibial surfaces for articulation with the corresponding condyles of the tibia and menisci.

These surfaces are separated from one another by the intercondyloid fossa and from the patellar surface by the intercondyloid fossa and from the patellar surface by faint grooves which extend obliquely across the two condyles. The latter groove is better marked; it runs lateralward to form a triangular depression.

The articular surface of the tibia consists of two facets, the central portions articulate with the condyles of the femur while their peripheral portions support the menisci of the knee joints which here intervene between the two bones. Between the two facets is the intercondyloid eminence surmounted on either side by a prominent tubercle, onto the sides of which the articular facets are prolonged. The lateral condyle

is usually somewhat smaller than the medial and is nearly circular in shape.

In the position of extension, the individual standing erect, the weight of the trunk is transmitted from the head of the femur in a vertical line which passes through the external condyle of the femur and tibia.

When full extension of the leg is reached and maintained the lateral part of the groove of the lateral condyle of the femur is pressed against the anterior part of the corresponding meniscus while the medial part of the groove rests on the articular margin in front of the lateral process of the tibial intercondyloid eminence. Into the groove on the medial condyle is fitted the anterior part of the medial meniscus, while the anterior crucial ligament and the articular margin in front of the medial process of the intercondyloid eminence are received into the forepart of the intercondyloid fossa of the femur.

In full flexion the posterior parts of the femoral condyles rest on the corresponding parts of the meniscotibial surfaces, and the semilunar area of the median condyle articulates with the median vertical facet of the patella. As the limb moves from flexion to extension the posterior two-thirds of the tibial articular surfaces of the two femoral condyles gradually shift forward, and the patella moves upward in the manner previously described.

In walking, greater weight is placed on the lateral condyle of the tibia than on the medial, and the inferior surface of the condyles articulates with the tibia. With these facts in mind, the results in the cases we studied may be detailed. The ages of the patients whose joints were examined, the number of joints at various age periods and the percentage of cases showing changes are recorded in Table I.

TABLE I

THE AGE DISTRIBUTION OF THE JOINTS EXAMINED AND THE NUMBER AND PERCENTAGE SHOWING CHANGES

Ages by Decades of Years	0 – 9	10 – 19	20 – 29	30 – 39	40 – 49	50 – 59	60 – 69	70 – 79	80+
Number of Joints	3	2	1	6	9	20	28	19	12
Percentage Showing Changes	0	0	0	66	100	95	100	94	91

All but six of the 100 joints were obtained from patients over 30 years of age and it is obvious from the table that anatomical changes are observed in the joints in increasing frequency with advancing age. By and large, there were very few knee joints which were absolutely normal after the age of 40 years. The areas of the joint showing changes are listed in Table II.

The patella showed changes in 81 cases. The commonest site was in the median horizontal pair of facets, and it was more common to observe them on the median than the lateral side. The corresponding articular surface of the femur showed changes more often than the condyles. In other words, the part of the patella coming in contact with the femur most frequently, as in walking, namely the median horizontal facets, is the area showing alterations in structure most commonly. The other parts of the patella which come in contact with the femur in extreme flexion and extension show changes less frequently. The character of these alterations varied more in degree than in kind. Thus simple fibrillation of the cartilage, loss of substance or complete absence of cartilage was observed. The articular area of the femur likewise showed areas of degeneration and they were usually opposite the changes on the patella. At the edges where the cartilage was destroyed there was evidence of attempts on the part of the cartilage to regenerate with the resulting heaping up and overgrowth of cartilage and bone.

TABLE II

SITE OF CHANGES OBSERVED IN 100 KNEE JOINTS

	Number of Joints Showing Changes
Patella	81
Interpatellar Groove	65
Median Condyle of Femur—Anterior	43
" " " " Posterior	7
Lateral Condyle of Femur—Anterior	36
" " " " Posterior	5
Median Condyle of Tibia	55
Lateral Condyle of Tibia	64

The inferior articular facets of the femoral condyles which articulate with the central portion of the tibial condyles showed lesions on the median side in 43 cases and on the lateral in 36. The corresponding articular surfaces of the tibia were involved in 55 and 64 cases respectively. In other words, the median femoral condyle was more often affected than the lateral, and the lateral condyle of the tibia more often than the medial. The posterior femoral condyles were altered seven times on the median side and five times on the lateral. The portion of the tibial condyles covered by the menisci did not show alterations or degeneration unless the menisci had been damaged and displaced.

It is noticeable and significant that the areas of the knee joint which were affected in these cases were those subjected to the greatest amount of weight and movement. The wear and tear of the tissue resulting from movement is apparent. It has already been stated that the patellar

femoral articulation is partly a gliding joint but inasmuch as the size, shape, and position of the patella and patellar surface of the femur vary in different individuals, the surfaces are not mutually adapted to each other. There can be no doubt that this lack of mutual adaptation causes wear and tear of the opposing surfaces with resulting tissue changes. It is perhaps significant that the tissue changes occurring with advancing age are most conspicuous over the areas of contact and, in so far as the patella and femur are concerned, over the areas which are subjected to most friction during movement.

I have already pointed out that the lateral condyle of the tibia showed alterations more often than the median, and that the part affected was the portion of the condyle uncovered by the semilunar cartilage. There is nothing remarkable about this when one remembers that in standing erect and in walking, the weight of the trunk is transmitted from the head of the femur in a vertical line which passes through the external condyle of the femur and tibia. In other words, the part of the tibia which supports the most weight and is in contact with the femur and unprotected by the meniscus is the one showing changes most often.

The other tibial facet and the articular surface likewise showed changes over the areas of contact.

Besides the erosions over the articular facets of the femoral condyles, areas of degeneration were observed on the lateral side of the median condyle. To explain the position of these erosions it is necessary to recall that in extension of the knee joint, the groove of the medial condyle is fitted into the anterior part of the medial meniscus, and the anterior crucial ligament and the articular margin in front of the medial process of the intercondyloid eminence are received into the forepart of the intercondyloid fossa of the femur. In some cases, the intercondyloid eminence is very prominent and comes in contact with the medial margin of the median condyle and in moving the knee these two surfaces are in contact. In every instance in which we have observed an erosion in this area, the median intercondyloid eminence has been high and in contact with this area. It is my belief, therefore, that these areas of degeneration result from the constant friction and contact with the two opposing surfaces.

Comment: From what has been said it is sufficiently clear that alterations in the knee joint increase with advancing age. It is likewise plain that the areas most frequently involved are the ones of contact and, therefore, those subjected to the greatest weight, movement and strain. The various hypotheses which have been advanced to explain these changes are not for consideration here. In view of our observations, however, there seems little escape from the conclusion that the anatomic changes are identical with those commonly recognized as occurring in degenerative or hypertrophic arthritis. In some cases they were seen in an early stage and in others in an advanced stage of degeneration. There is justification for the belief then that degenerative or hypertrophic arthritis is a process associated with aging of the joint tissues. A full explanation of the various factors which are responsible for this process is still awaited. However, the conception that aging of joint tissue contributes to the changes is essential for a complete understanding and evaluation of the clinical condition known as degenerative arthritis. All of the lesions in a joint involved with degenerative (hypertrophic) may be exaggerated or increased by trauma, hemorrhage, infection, the deposits of urates, the formation of loose bodies or static deformities. The final result, therefore, will depend upon the summation of a number of factors.

RECENT STUDIES IN RHEUMATOID (CHRONIC INFECTIOUS, ATROPHIC) ARTHRITIS*

BY M. H. DAWSON, M.D.,† AND R. H. BOOTS, M.D.†

IT is with a certain feeling of embarrassment that anyone from another city should accept an invitation from the Boston Orthopedic Club to discuss the subject of chronic arthritis. In New York we are all deeply conscious of the many contributions to the knowledge of this group of diseases which have been made by members of your Society and your stimulation stands foremost in our minds. On the other hand, the approach to the problem among the investigators of this city has been in large part determined by those interested in orthopedics while a considerable proportion of the work done in New York has been carried out by those interested in medical and bacteriological problems.

Originally, we were asked for a discussion of

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The Arthritis Clinic of the Presbyterian Hospital has adopted the terminology and classification of rheumatic diseases advocated by the British Ministry of Health and the International League for the Control of Rheumatism. In this classification the term rheumatoid arthritis is considered to be synonymous with the terms chronic infectious arthritis, atrophic arthritis, proliferative arthritis and primary progressive polyarthritis. The term osteoarthritis is used synonymously with the terms hypertrophic arthritis and degenerative arthritis.

Presented before the Boston Orthopedic Society at the November, 1932 meeting. Paper read by Dr. Boots.

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the infective hypothesis of chronic arthritis. We do not hold such an hypothesis. The term chronic arthritis is used to designate a whole group of diseases, some of which are undoubtedly associated with infection, while others are simply the result of age-period degenerative changes. Furthermore, chronic arthritis usually occurs as but one manifestation of a generalized disease process and its causation is the same as that of the disease of which it is a part. This is certainly true of gonorrhea, tuberculosis, syphilis, dysentery and gout. So also in those two great groups of chronic multiple arthritis variously designated by the terms rheumatoid, chronic infectious or atrophic and osteo, hypertrophic or degenerative, the joint manifestations are usually only one part of the general picture. We do believe, however, that the factor of infection plays a predominant rôle in the production of the symptom-complex known as rheumatoid (chronic infectious, atrophic) arthritis. Osteo (hypertrophic, degenerative) arthritis, on the other hand, may be considered as largely a manifestation of age-period changes. It may be expressed as the summation of joint insults due to many causes, e.g., metabolic, circulatory, endocrine and traumatic, occurring during the course of the patient's lifetime. It is also realized that the changes initiated by infection may be followed by secondary degenerative alterations in such a way that the eventual picture may closely resemble that of osteoarthritis.

Rheumatoid or atrophic arthritis must still be considered as a disease of unknown etiology. However, it presents many of the characteristics of an infectious process. There is frequently a preceding history of acute upper respiratory tract infection, common cold, pharyngitis, tonsillitis, quinsy or sinusitis. A low grade fever accompanied by a slight leucocytosis is a common manifestation of the disease. Frequently the pulse is rapid and a definite degree of anemia occurs in over 80 per cent of cases. Muscular atrophy occurs to a degree too considerable to be ascribed to mere disuse. The patient almost invariably loses weight and appears chronically ill. All these clinical features are consistent with infection. Until recently, the evidence that rheumatoid arthritis was related or due to infections was purely clinical. In the past few years, however, certain bacteriological and pathological investigations have served to lend considerable support to the infectious theory of the etiology of the disease. It is concerning these investigations that the present paper is concerned.

BACTERIOLOGICAL STUDIES

Among those who hold that rheumatoid arthritis results from infection, two viewpoints are popular; the one, that the disease is caused by the organisms actually growing in the affected tissues; the other, that the disease represents the reaction of the individual to some focus of infection without the organisms which cause the infection actually gaining access to the joints or tissues. This response of the affected parts may be in the nature of a hypersensitive or allergic phenomenon, or it may be due to a simple toxic reaction to noxious products absorbed from the primary focus.

Evidence in support of the belief that organisms are actually present in the joints and tissues of patients suffering from rheumatoid arthritis has not been convincing. Recently interest in this phase of the problem has been revived by the work of Cecil, Nicholls and Stainsby[1]. By the use of a special technique these investigators claim to have demonstrated the presence of streptococci in the blood stream of 68 per cent of a series of cases examined. Similar results have been reported by Gray and Gowen[2]. However, a large number of other investigators have reported negative findings[3]. The results of our own investigations in this field have been recently published[4]. Our findings may be summarized as follows: (1) Blood cultures on patients suffering from rheumatoid arthritis, carried out according to the technique of Cecil, Nicholls and Stainsby, failed to yield organisms that could be considered of etiological significance; (2) control material subjected to the same manipulations yielded growth in approximately the same proportion of cases. (See Fig. 1.)

Following the failure to confirm the cultural work of Cecil and his co-workers an extensive investigation was made of agglutination reactions in rheumatoid arthritis. Nicholls and Stainsby[5] had previously reported that the sera of rheumatoid arthritis patients possessed the capacity of agglutinating their "typical strains" of streptococci in high dilutions. Through the courtesy of these workers a number of their strains were made available for the study. In addition to these strains a large number of strains of other Gram positive cocci were similarly employed in agglutination studies. A report of these investigations has been recently published[6]. The findings may be briefly summarized as follows:

In the majority of cases the sera of patients with rheumatoid arthritis possess the property of agglutinating strains of Streptococcus hemolyticus (at 55° c.) to an extraordinarily high titer. No correlation was observed between the source of the strains and their agglutinability in the sera examined. Strains of Streptococcus hemolyticus obtained from cases of scarlet fever, erysipelas and rheumatic fever were agglutinated to as high a titer as were the "typical

strains" of Cecil, Nicholls and Stainsby. Agglutination tests with a wide variety of other gram-positive cocci revealed that these organisms were either not agglutinated at all or were agglutinated to a very low titer. An exception to this generalization is made in the case of R pneumococci which were agglutinated to almost as high a titer as were strains of Streptococcus hemolyticus. With the exception of certain instances, control sera did not agglutinate strains of Streptococcus hemolyticus in a titer which was comparable with that observed in rheumatoid arthritis.

It has been further established that the property of rheumatoid arthritis serum responsible for the agglutination of hemolytic streptococci is definitely related to the duration of the disease and the age of the patient. The phenomenon is not present until the disease has been well established in its classical form for several months and the sera of young patients do not agglutinate in significantly high dilutions. In no instance did the serum of a patient suffering from osteo (hypertrophic) arthritis give a positive agglutination.

In the present state of knowledge it is difficult to evaluate the significance of the agglutination reaction in rheumatoid arthritis. The findings suggest that infection by Streptococcus hemolyticus may play a rôle in the production of the disease but further work is required to establish this hypothesis.

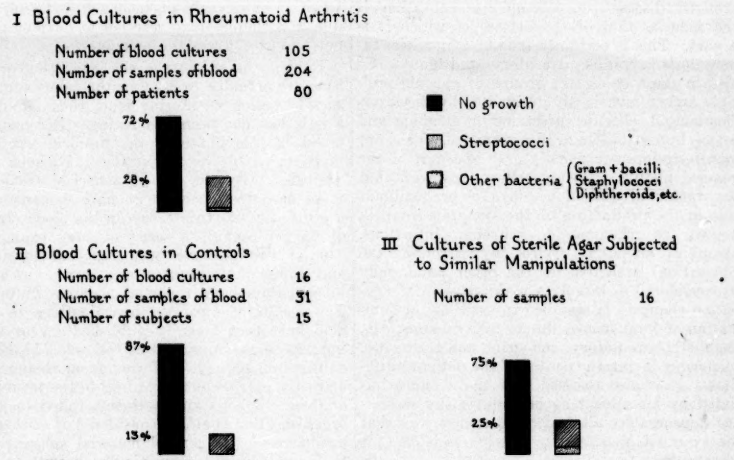

FIGURE 1.

SUBCUTANEOUS NODULES

In the Arthritis Clinic of the Presbyterian Hospital we have been interested in observing the frequency and character of subcutaneous nodules which occur in patients suffering from rheumatoid arthritis. Despite the fact that such nodules constitute an even more prominent feature of the disease than do the corresponding subcutaneous lesions in rheumatic fever considerably less attention has been paid to their occurrence. The subcutaneous nodule of rheumatic fever is generally regarded as a typical and highly characteristic lesion and is considered by almost all pathologists as "morphologically and genetically essentially the same as the Aschoff body in the myocardium". We were, therefore, anxious to study the nodules in rheumatoid arthritis both pathologically and bacteriologically.

Subcutaneous nodules have been observed in a total of 72 patients. This represents an incidence of approximately 20 per cent. They have only been observed in cases of rheumatoid arthritis. In spite of diligent search we have never observed such nodules in cases of osteo (hypertrophic) arthritis. These nodules are usually considerably larger than those observed in rheumatic fever, the larger ones frequently attaining a diameter of one to two centimeters. The most frequent situation is in the region of the olecranon process, but they have also been observed over the dorsum of the hand, the scalp, the patella and in the region of the sacrum. The lesions are located in the subcutaneous tissues and not infrequently appear to bear a relation to tendon sheaths and the walls of bursae. They usually develop slowly and rarely cause pain. The typical gross appearance of the nodules is seen in figure 2.

Preliminary pathological studies on the structure of these lesions have been published elsewhere[8]. It is sufficient at this time to state that the histological appearance of the nodules is highly characteristic and that they show a

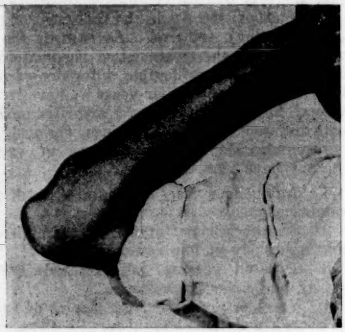

FIGURE 2.

striking resemblance to the nodules which occur in rheumatic fever. The essential features of the lesion consist of a central area of necrosis, a surrounding zone of large mononuclear cells arranged in characteristic radial fashion and an enveloping zone of dense and relatively avascu-

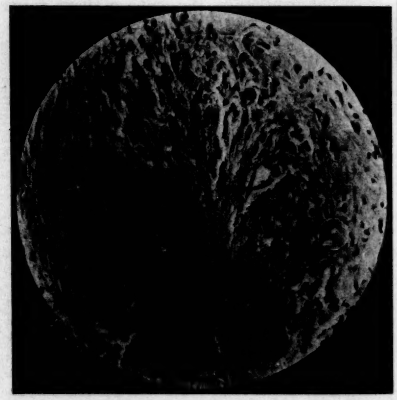

FIGURE 3.

lar fibrous connective tissue. A typical section is shown in figure 3. Notwithstanding the fact that bacteriological studies on these nodules have been entirely negative, their general appearance is distinctly that of an inflammatory granuloma and their structure is highly suggestive of an infectious process.

SEDIMENTATION RATE OF THE ERYTHROCYTES

During the past three years the determination of the sedimentation rate of the erythrocytes has been carried out as a routine procedure in our clinic[9]. The test is very simply performed and gives information of distinct clinical value. Westergren's modification of Fahraeus' technique has been employed. The results of over 2000 observations on several hundred patients may be summarized as follows:

In active cases of rheumatoid arthritis the sedimentation rate of the red blood cells is, as a rule, greatly elevated, usually attaining values exceeding 30 mm. in one hour. The rate parallels to an extraordinary degree the severity

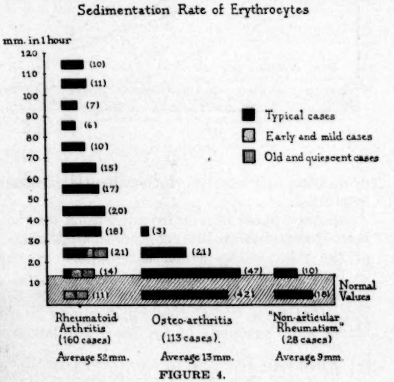

FIGURE 4.

and extent of the arthritis process. As a patient becomes worse the rate increases and with improvement it tends to return to normal values. On the other hand, in cases of osteo (hypertrophic) arthritis, the sedimentation rate, while occasionally slightly elevated, rarely attains values greater than 20 or 30 mm. in one hour. The test is therefore of assistance in the differentiation of the two diseases, rheumatoid and osteo-arthritis. It should be pointed out, however, that the rate is also definitely accelerated in other forms of arthritis associated with infection such as tuberculosis, gonorrhea and in acute gout. Cases of myositis, neuritis and fibrositis have all shown a normal sedimentation rate.

In figure 4 there is presented a summary of our observations in cases of arthritis and non-articular rheumatism.

In figures 5 and 6 there is shown a series of observations of the S.R. on patients over a two year period. Figure 5 represents determinations

on a series of five patients who showed distinct clinical improvement. Figure 6 shows similar observations on five other patients, in whom the disease continued in an active form. The test, therefore, offers a convenient method of following the degree of activity of the process and

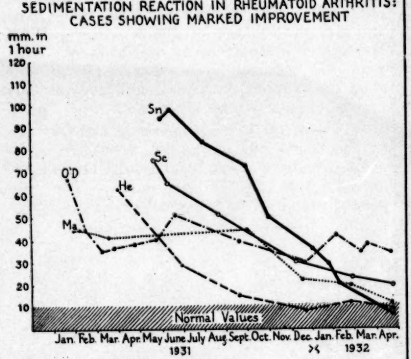

FIGURE 5.

evaluating the results of therapeutic measures instituted.

Another phase of our investigations on rheumatoid arthritis has been concerned with a study of the relationship which this disease appears to bear to orthodox rheumatic fever. Considerable evidence has been assembled which suggests that these two clinical entities may be different manifestations of the same fundamental

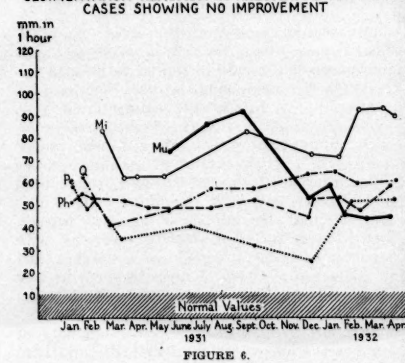

FIGURE 6.

disease process. However, in the present stage of the study such a conception can only be advanced as an hypothesis.

VACCINE THERAPY

Before concluding, brief mention will be made of our experience with one particular form of therapy, the use of vaccines in the treatment of chronic arthritis. In the first place, we feel that there is no justification whatever for the routine use of vaccines in the treatment of osteo (hypertrophic) arthritis. As regards the use of vaccines in the treatment of rheumatoid arthritis there is considerable difference of opinion. During the past four years we have treated several hundred rheumatoid arthritis patients with different forms of vaccines, variously administered. The following forms of vaccine therapy have been employed:

1. Autogenous vaccines administered subcutaneously.
2. Streptococcus "antigens" administered subcutaneously and intravenously.
3. Typhoid vaccines administered intravenously and subcutaneously.
4. Vaccines prepared from strains of Streptococcus hemolyticus administered both subcutaneously and intravenously.

In the above procedures both small and large doses have been employed. In addition a considerable series of patients has been given weekly injections of minute amounts of isotonic sodium citrate for control purposes.

The results of the foregoing studies may be summarized as follows:

In our experience, the value of these forms of vaccine therapy in the treatment of rheumatoid arthritis remains unproved. In particular it has been found impossible to influence favorably the sedimentation rate of the erythrocytes in any convincing proportion of cases. Patients on the other forms of treatment than vaccine therapy show equally as great clinical improvement as those on vaccine therapy. It is our feeling that the results of vaccine therapy vary with the enthusiasm of the user.

SUMMARY

The results of our studies on rheumatoid arthritis may be summarized as follows:

1. Contrary to the results of certain other investigators, we have not been able to recover streptococci from the blood or tissues of patients suffering from this disease.
2. In the majority of cases the sera of patients with rheumatoid arthritis possess the capacity of agglutinating strains of *Streptococcus hemolyticus* (at 55° c.) to an extraordinarily high titer. This is a highly characteristic phenomenon, but further work is required before conclusions can be drawn as to its significance.
3. Subcutaneous nodules, which show a striking histological resemblance to those occurring

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in rheumatic fever, have been observed in approximately 20 per cent of rheumatoid arthritis patients.

4. The sedimentation rate of the erythrocytes in rheumatoid arthritis parallels to an extraordinary degree the severity and extent of the arthritic process. The test offers a convenient method of evaluating the results of therapeutic measures and is useful as an aid in differentiating rheumatoid and osteo-arthritis.

5. It is our belief that while vaccine therapy may be accorded a trial in the treatment of rheumatoid arthritis, its value has not been proved. There seems to us no justification for the use of vaccines in the treatment of osteo-arthritis.

6. Pathological and immunological evidence confirms the clinical impression that rheumatoid arthritis is a clinical entity and suggests that it is infectious in origin.

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SPECULATIONS ON THE ETIOLOGY OF RHEUMATOID ARTHRITIS*

Based on Physiological Studies of Normal Joints

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Mr. President and Members of the Boston Orthopedic Club:

I GREATLY appreciate being allowed to address your society because the experimental work to be presented has been carried out under the auspices of a fund established by friends and patients, under the direction of Dr. Frank Ober, in memory of one of your former distinguished members, the late Dr. Robert W. Lovett.

Rheumatoid arthritis is a disease of great social and economic importance because hundreds of thousands of people suffer from it and many of these sufferers are partially or totally incapacitated. Man has had to contend with this disease for centuries, physicians have treated it for centuries, many forms of therapy are advocated and employed, yet the results obtained are at times anything but gratifying and not infrequently the disease progresses despite all forms of treatment. What can be the answer to all this? There is only one answer and it is that the exact etiology of the disease remains unknown. Further evidence that the exact etiology is unknown is furnished each year or so when another fund is established or a grant made for the study of arthritis in one of our hospitals or medical centers. The fact that the exact cause is still unknown is no reason for us to be discouraged. It only means that each worker engaged in studying this disease and each practitioner of medicine who treats these patients should study them more carefully and thoroughly than ever, correcting all abnormalities, treating the patient as a whole and observing the effect on the course of the disease. This is the only way we shall contribute further to our knowledge and understanding of this disease and eventually solve the riddle.

A concise description of the disease will enable us to understand what we are confronted with in each case of rheumatoid arthritis. The onset may be gradual or sudden. Constitutional symptoms usually precede those referable to the skeletal system. It does not respect age, sex, race, or social position, although it occurs more often in the second, third and fourth decades of life, women are more frequently affected than men, white oftener than colored and the poor more often than the rich. Heredity is probably a factor. The individual affected is usually the asthenic type, whose symptoms include weakness,

*Other terms used to designate rheumatoid arthritis are (1) proliferative arthritis, (2) atrophic arthritis, (3) Type I arthritis.

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easy fatigability, loss of appetite, various gastrointestinal complaints, insomnia, loss of weight, etc. On physical examination one is apt to find an asthenic individual, showing pallor as well as evidence of slight anemia, underdevelopment, undernourishment, various trophic changes of the skin and nails, muscle atrophy, frequently marked signs of increased vasomotor activity as well as joint changes. Generalized lymphadenopathy and splenomegalia have been observed. There may or may not be obvious foci of infection. Slight fever, leukocytosis, an increased sedimentation rate of the red blood corpuscles and positive agglutination tests against certain strains of streptococci are frequently observed and suggest infection. Joint symptoms may be present before there are obvious signs of joint involvement. The joint symptoms may be migratory. One sees the monarticular form but the polyarticular form is the more common. The smaller joints are usually affected first and the involvement is generally bilateral. The first joint symptoms may be only stiffness and aching, rarely one sees a patient who never has true pain. As a rule, the symptoms of stiffness and aching are followed by swelling, redness, heat and pain in one or more joints. Effusion into the joint may or may not be present. Rarely one observes progressive limitation of motion and gradual increase in joint ankylosis with very few joint symptoms. X-ray examination early in the disease may show no deviation from the normal, later one finds soft tissue swelling, narrowing of the joint space, destruction of the articular cartilage and rarefaction of the subchondral bone.

One of the most important things to be remembered concerning rheumatoid arthritis is that the disease is characterized by remissions and relapses. Therefore, one often finds it difficult to evaluate the form of therapy employed because he is always confronted with the question: Is the improvement due to the treatment employed or are we dealing with a natural remission occurring during the time a specific form of therapy was used? During a remission, the patient may become absolutely symptom-free, but as a rule there are certain residual aches and pains. Many factors would seem to play a rôle in precipitating a relapse or bringing about a remission. For example, during the state of pregnancy, in one patient one may observe a remission, in another a relapse. Remissions may last months or years. In the progressive form of the disease, each relapse leaves more evidence of crippling and deformity. Because we are dealing with a disease characterized by remissions, we should be more cautious in speaking of cures. Before stating one has cured a patient with rheumatoid arthritis, he must be absolutely certain that his diagnosis was correct. There are many other diseases involving the joints which are very often mistaken for rheumatoid arthritis. Granted that the diagnosis is correct, one should allow for a lapse of at least five years, preferably ten, before he pronounces a patient cured.

Considering the number of patients who suffer from this disease, it is surprising that the medical literature contains so little information concerning the general postmortem findings in cases of rheumatoid arthritis. More information of this sort, particularly in cases which have been very carefully studied, would undoubtedly add considerable material to our total knowledge of the disease. The pathology of the joints proper has been studied quite extensively, particularly the well-advanced lesions and the end stages and have been described in great detail in the exhaustive monograph of Nichols and Richardson[1]. The earlier changes are perhaps better described in a very excellent paper by Hare[2]. These changes consist of proliferation of the synovial membrane and connective tissue of the subchondral bone spaces, round cell infiltration, increased vascularity and bone atrophy. The most marked and constant feature of the joints is that of proliferation, all other changes are for the most part secondary to it. Proliferation of the synovial membrane and of the connective tissue of the subchondral bone spaces is usually present. Destruction of the articular cartilage results if the proliferating synovial membrane called pannus grows over and eventually invades it. Invasion of the articular cartilage from below similarly results because of connective tissue proliferation in the subchondral bone spaces. These changes have been interpreted by some workers as being due to infection. Dr. Boots has discussed this aspect, therefore we shall confine our remarks to other possibilities.

It has been our belief and hope that if we could add to our knowledge concerning the anatomy and physiology of normal joints, we might be in a better position to understand how the above-mentioned proliferative changes are brought about. In other words, is it possible to produce these proliferative changes by simply interfering with or altering the physiology of a normal joint? If so, then we would be in a better position to evaluate some of the current theories concerning the etiology of rheumatoid arthritis. Therefore, it might be well to briefly review the anatomy of normal joints as well as to present certain fundamental physiological facts in the case of normal joints. Later, we shall attempt to speculate as to how proliferation of the synovial membrane might be produced.

Most joints are relatively simple structures, consisting of two bones, the cartilaginous ends of which are in apposition one with another. The cavity is lined by synovial membrane which is nothing more than connective tissue which varies with the tissue it overlies. Therefore, in a joint we have the areolar tissue type, the adi-

pose type and the fibrous tissue type of synovial membrane. The synovial membrane can be removed from a normal rabbit's knee joint and a few months later one finds a re-formed synovial membrane which is almost indistinguishable from that removed[3]. This experiment definitely proves that synovial membrane is not a highly specialized type of membrane as some workers have contended. These simple types of connective tissue are the ones which proliferate readily.

Underneath the synovial membrane one finds a very rich network of blood vessels. Whether this subsynovial blood supply represents an end type of circulation or one having a free collateral system is a point which remains unsettled. It will be pointed out later why further information concerning this point is necessary in order to better understand what rôle interference with circulation to a joint might play in the production of joint changes. Besides the rich subsynovial vascular supply, each joint has a similar system of subsynovial lymphatics. Nerves and pacinian corpuscles have been demonstrated in the subsynovial tissues. The cartilage of joints is hyaline in nature. The cartilage at the perichondrial margins probably receives its nourishment from the circulus articuli vasculosus of Hunter. It is in this region that articular cartilage shows the greatest ability to repair. The central portion of the articular cartilage is avascular and the question of how it obtains its nourishment is not definitely known. It may be that it is derived solely from the synovial fluid or from plasma water diffusing through from the subchondral bone vessels, or a combination of the two. We do know that the ability of the central portion of articular cartilage to regenerate is at best very feeble and limited. This is true whether cartilage defects are produced surgically or are due to repeated trauma[4, 5].

All joints contain synovial fluid. Various theories have been proposed concerning its origin and nature. These are as follows:

1. Normal synovial fluid represents the products of disintegration and degeneration of normal joint tissues brought about by motion.
2. Normal synovial fluid is a transudate from blood or lymph.
3. Normal synovial fluid is a transudate from blood and lymph containing the products of disintegration and degeneration of normal joint tissues.
4. Normal synovial fluid is a secretion of the synovial membrane lining cells.
5. Normal synovial fluid is secreted by special cells of the synovial membrane.

No one of these theories has gained general acceptance because the known physical and chemical properties of normal synovial fluid have been insufficient to allow one to define it chemically or to speak with certainty concerning its origin.

The astragalotibial joint of normal young cattle is a source of a large quantity of easily obtainable synovial fluid. Therefore, we have made a very complete chemical analysis of simultaneously obtained synovial fluid and arterial blood from twenty-four young cattle in order to make a comparison of these two body fluids. From these studies it was learned that synovial fluid is a clear, viscid liquid which contains on an average 132 nucleated cells per cubic millimeter of which 94 per cent are phagocytic cells and 6 per cent non-phagocytic cells. Normally, red blood cells are not present; if found, they are due to trauma[6]. The following determinations have been made on both blood and synovial fluid: water content, pH, total solids, specific gravity, freezing point, osmotic pressure, total nitrogen, mucin, total protein, cholesterol, non-protein nitrogen, urea, uric acid, sugar, chlorides, phosphorus, CO_2 content, lactic acid, calcium, sodium, and total base[7].

If one compares the concentration of a non-electrolyte such as urea in the synovial fluid and arterial serum, he finds that they are practically identical. For this reason and others based on the data obtained in this study, we believe that synovial fluid is probably a simple diffusate coming from the rich, subsynovial vascular supply and is not a product of disintegration and degeneration, a diffusate from lymph, or a secretion of special synovial lining cells or ordinary synovial lining cells. Cross sections from a dog's knee joint in which the vessels were injected with a form of graphite called Hydrokollag 300 show very clearly that the subsynovial vessels in many instances are separated from the joint cavity by only a few layers of cells whereas in others six to twenty cells intervene between the lining membrane and the underlying vessels. With this visual picture in mind, it is not very difficult to understand why normal synovial fluid is probably nothing more than plasma water containing mucin and a minimal amount of protein.

Granted that normal synovial fluid is a simple diffusate, one must next consider how interchange takes place. We know from previous experimental work[8, 9, 10, 11] that true solutions can gain entrance into the joint from the underlying blood vessels and similarly that true solutions leave the joint very rapidly and enter the subsynovial blood vessels and lymphatics.

However, the information concerning the manner in which proteins are removed from joints as well as the rate of removal has, in so far as we could learn from the literature, never been determined. The experiments which we are reporting were performed on the knee joints

of normal adult dogs and have been published elsewhere[12]. The proteins used in these experiments were those contained in egg white and horse serum. In each experiment the protein was injected directly into the knee joint and its appearance in the blood stream and lymphatic juice was determined by precipitin tests done at frequent intervals using the serum of rabbits immunized against the particular protein employed.

If egg white is injected into the knee joint of a normal dog, it can be detected in the blood stream as early as 15 to 20 minutes following the injection. If the knee joint is passively exercised by means of a mechanical rotating device, the proteins are removed at a much faster rate and in much larger quantities. (See table I.)

RESULTS OF PRECIPITIN REACTIONS

Time After Injection	Dilution of Serum Positive	
	Unexercised Dog	Exercised Dog
Control	1/2	1/2
10 min.	1/4	1/4
25 "	1/4	1/8
45 "	1/8	1/16
1 hr.	Lost	1/32
2 hrs.	1/4	1/32
3 "	1/2	Not done

TABLE I. EXPERIMENT I. The concentration of egg white in the blood stream following injection into the knee joint of 3 cc., showing the relative absorption from passively exercised and unexercised joints.

In order to determine whether the proteins are removed from the joint by way of the lymphatic or vascular systems, the following experiment was performed. Two normal dogs were used. Under anesthesia all of the lymphatics emptying into the great vessels of the neck on the right and left sides were tied off before the experiment was begun, thus eliminating all communications between the lymphatic and vascular systems. One dog was kept absolutely quiet whereas the leg of the second dog was constantly exercised by means of the same mechanical rotating device. Cannulae were inserted into the thoracic ducts of both dogs in order to allow us to collect frequent specimens of thoracic duct lymph. Blood samples from each dog were also taken at the same intervals, thus enabling us to determine whether any protein can gain entrance into the blood stream if the lymphatics entering the great vessels of the neck are completely tied off. From Table II it will be seen that the control blood sample showed no precipitin reaction, but what is of more interest is that no positive precipitin tests for egg white were obtained from any of the blood samples taken throughout the entire length of the experiment, showing conclusively that proteins are unable to gain entrance into the blood stream if the lymphatic vessels are tied off. In the case of the exercised dog it will be seen that the egg white was detectable in the thoracic duct lymph at the end of one-half hour. At the end of an hour it could be detected in a dilution of 1/256, whereas at the end of the second hour it was detected in a dilution of 1/512. In the case of the unexercised dog, the leg was left absolutely quiet, not even massaged, and it is readily seen that none of the egg white was detectable in the thoracic duct lymph at the end of three hours. From this type of experiment we feel we have the right to conclude that proteins such as are contained in egg white are removed from the joint only by way of the lymphatics and if all communications between the vascular and lymphatic systems are eliminated, none of the injected proteins can gain entrance into the blood stream. We further believe that this type of experiment demonstrates very clearly the value of exercise in the removal of proteins from the joint.

RESULTS OF PRECIPITIN REACTIONS

Time After Injection	Unexercised Dog		Exercised Dog	
	Dilutions Positive		Dilutions Positive	
	Serum	Lymph	Serum	Lymph
Control	—	—	—	—
10 min.	—	—	—	—
20 "	—	—	—	—
30 "	—	—	—	1/16
45 "	—	—	—	1/128
1 hr.	—	—	—	1/256
2 hrs.	—	—	—	1/512
3 "	—	—	Not done	Not done

TABLE II. EXPERIMENT IV. The concentration of egg white in the blood stream and thoracic duct lymph following injection into knee joint of 3 cc. In this experiment, the leg of the unexercised dog *was not massaged.*

Table III represents an experiment similar to the preceding one. All communications between the lymphatic and vascular systems were severed. Samples of blood and thoracic duct lymph were collected at the same intervals. The knee joint of one dog was again kept constantly exercised, whereas the knee joint of the other dog was kept quiet but the muscles of the leg and foreleg were constantly massaged. The results are similar to those in the preceding experiment. Again the proteins were not detected in the blood stream of either dog in greater concentration than in the control samples. It will be seen that the appearance time of the proteins in the thoracic duct lymph in the exercised dog is almost a duplicate of the preceding experiment. However, in the case of the unexercised dog in which the leg was constantly massaged the proteins were detectable

in dilutions of 1/128 at the end of the second and third hours. Therefore, this experiment not only confirms the findings in the preceding experiment but also demonstrates that the removal of protein from a joint is greatly enhanced if the muscles are constantly massaged.

RESULTS OF PRECIPITIN REACTIONS

Time After Injection	Unexercised Dog		Exercised Dog	
	Dilutions Positive		Dilutions Positive	
	Serum	Lymph	Serum	Lymph
Control	1/8	1/2	1/4	1/4
10 min.	1/4	1/2	1/4	1/2
30 "	1/4	1/4	1/8	1/32
1 hr.	1/4	1/32	1/8	1/256
2 hrs.	1/4	1/128	1/4	1/512
3 "	1/4	1/128	Not done	Not done

TABLE III. EXPERIMENT V. The concentration of egg white in the blood stream and thoracic duct lymph following injection into the knee joint of 3 cc. In this case, the leg of the unexercised dog *was massaged.*

In the next set of experiments, we were interested in finding out whether there is any difference in the removal of serum albumin as compared with serum globulin. Therefore, in each instance horse serum rather than egg white was injected into the knee joints of both dogs. Again, all the lymphatics entering the great vessels of the neck were tied and lymph was collected from the thoracic duct. Rabbits had been previously immunized against the serum albumin and serum globulin fractions of horse serum, thus enabling us to carry out precipitin tests at frequent intervals on the thoracic duct lymph and blood for both serum albumin and serum globulin. From Table IV it will be seen that in the case of the exercised dog albumin could be detected in the thoracic duct lymph at the end of one-half hour in a dilution of 1/2048 and that it remained at this concentration in the lymph up to three hours. However, one notes that in the same exercised dog very little if any of the horse serum globulin had been removed from the joint at the end of three hours. In the case of the unexercised dog it will be seen that albumin was not detected in a dilution of 1/2048 in the thoracic duct lymph until two hours had passed. Again we see that very little if any globulin had been removed from the unexercised dog at the end of three hours. These experiments give us further evidence that exercise greatly increases the rate of removal of proteins from normal joints. They also tell us that the larger protein molecule, globulin, is removed from the joint with great difficulty whether the joint is exercised or unexercised. Thus we can state definitely that the size of a molecule which can be removed from a joint with great ease, lies somewhere between a molecular weight of 72,000 and 175,000.

This type of experiment not only allows us to state that the proteins contained in normal synovial fluid are removed only by way of the lymphatics but also gives us definite information concerning the size of a particle which can readily pass through the synovial membrane. Information of this sort should add to our understanding of the mechanism of joint effusions.

RESULTS OF PRECIPITIN REACTIONS

Immune Serum Used and Time After Injection	Dilutions Positive			
	Serum		Lymph	
	Unexercised	Exercised	Unexercised	Exercised
Anti-globulin				
Control	1/256	1/256	1/128	1/128
10 min.	1/256	1/256	1/256	1/256
30 "	1/256	1/256	1/256	1/256
1 hr.	1/256	1/256	1/256	1/128
2 hrs.	1/256	1/256	1/256	1/128
3 "	1/256	1/256	1/256	1/128
Anti-albumin				
Control	Negative	Negative	Negative	Negative
10 min.	"	"	"	"
30 "	"	"	"	1/2,048
1 hr.	"	"	1/8	1/2,048
2 hrs.	"	"	1/2,048	1/2,048
3 hrs.	"	"	1/2,048	1/2,048

TABLE IV. EXPERIMENT VI. The concentrations of albumin and globulin fractions of horse serum in the blood stream and thoracic duct lymph after injection of 3 cc. undiluted horse serum.

Therefore, it would seem that a joint effusion may result from too rapid a diffusion of the plasma filtrate from the subsynovial blood vessels into the joint cavity or from interference with the removal of proteins, or from a combination of these two factors. Therefore, increased permeability of these vessels, no matter what the causative agent might be, would result in a more rapid formation of synovial fluid which is much richer in protein than normal synovial fluid. The increased protein concentration of the synovial fluid would naturally attract more water because its presence greatly increases the osmotic pressure. Therefore, if proteins are not removed from the joint as rapidly as they gain entrance a joint effusion would result and be maintained. These experiments suggest that simple tests might be devised which would enable us to state with some degree of certainty why an effusion has resulted and why it persists.

What application can be made of the above experimental data in a discussion of the etiology of rheumatoid arthritis? It would appear to allow for a certain amount of speculation not necessarily of the wildest sort. In rheumatoid arthritis the one most important finding is proliferation of the synovial membrane. This process is responsible for the marked joint changes we observe in this disease including the

destruction of cartilage and the fibrous ankylosis. What causes the synovial membrane to proliferate? Synovial membrane as we have already stated is composed of a simple type of connective tissue, the type which proliferates most readily. What stimulus is necessary for such proliferation? Is infection the only factor which brings about such changes? Direct injury to the synovial membrane, an intra-articular allergic reaction, as well as infection, might very likely be sufficient stimulus to cause such proliferation. Any one of these three factors as well as others might be associated with an increased permeability of the subsynovial blood vessels of a degree sufficient to allow for the formation of a joint effusion. It may be reasoned that accompanying fibrosis of the synovial membrane and subsynovial tissue could interfere with the removal of proteins from the joint and thus give rise to a joint effusion or at least delay its removal once it has formed. Given a joint effusion, we have an ideal culture medium for connective tissue growth because it has a high protein content, contains fibrin and all the constituents of serum. Since the long-continued presence of fibrin elsewhere in the body in other disease processes is almost always followed by proliferation of fibroblasts and vascular endothelium, one might reason that such a sequence might very well follow in cases of long-standing joint effusion. Is a joint effusion a prerequisite for the proliferative process? Clinical observations would tend to answer this question in the negative, but can we necessarily say that an increase in synovial fluid has not existed at some time?

Can vascular impairment of itself produce the intra-articular changes of rheumatoid arthritis? Mention is frequently made of the changes which result because of the poor peripheral circulation[8, 15]. We have considerable evidence in certain of these patients, particularly the progressive type, of such vascular impairment. The evidence in such cases consists of signs of increased vasomotor activity, cold, clammy cyanotic extremities with reduced skin temperature, decreased circulation as observed in the reduction in the number of visible nail bed capillaries and abnormal arterial and venous sugar curves[13, 14]. If the circulation to a joint does not have an extensive collateral circulation, then it is reasonable to suppose that the normal physiology of the joint might well be interfered with. These possibilities all lend themselves to experimentation and are some of the problems we are undertaking. Certainly we know that any form of therapy resulting in vasodilatation gives an arthritic considerable relief, sometimes it is almost miraculous even though it lasts but a few days.

There are other theories which have been proposed concerning the etiology of arthritis which are more difficult of interpretation, requiring considerable imagination in order to see how they could produce intra-articular changes. One such theory is that certain cases of arthritis are related to hyperparathyroidism. This has been proposed by Funsten[16] and Ballin[17].

Hyperparathyroidism or generalized osteitis fibrosa cystica is now conceded to be a clear-cut, distinct disease entity caused by an excessive secretion of the parathyroid hormone. It was first produced experimentally by Collip[18]. A year or so later (1926), it was first recognized in man, by Mandl[19] in Vienna and by DuBois[20] in America. This disease is characterized by very definite symptoms and signs as well as alterations in the calcium and phosphorus metabolism. These symptoms and signs are

1. Polyuria
2. Polydipsia
3. Weakness and loss of strength
4. Constipation
5. Loss of appetite
6. Loss of weight
7. Indefinite muscle and joint aches and pains (commonly diagnosed rheumatism, arthritis, or neuritis)
8. Bone tenderness
9. Frequent fractures following very slight trauma
10. Decreased excitability of the nerves
11. Skeletal shortening
12. Kyphosis
13. Bone tumors
14. Kidney or ureteral stones (usually bilateral)
15. Characteristic x-ray findings (generalized decalcification, bone tumors and multiple bone cysts, etc.)
16. Anemia with leukopenia.

The alterations in the calcium and phosphorus metabolism are

1. An elevated serum calcium
2. A lowered serum phosphorus
3. An increased calcium excretion—urinary
4. An increased phosphorus excretion—urinary.

Funsten and Ballin reported no metabolism studies. Funsten stated that in suspected cases one should do repeated serum calcium determinations. By doing so, one would obtain one or two serum calcium values which were distinctly elevated, thus enabling him to make a diagnosis of hyperparathyroidism. All cases of hyperparathyroidism which we have observed have had a permanently elevated serum calcium and not transitory elevations such as reported by Dr. Funsten. We usually ascribe transitory elevations in serum calcium as being due to a failure to obtain a fasting blood sample, and if this has not been the case, we always raise the question of whether there has been any error in technique. It must be remembered that a

serum calcium determination tells one only the height of the calcium stream and not its direction of flow into the bones or into the excretory channels. Thus one can understand the need of doing total calcium and phosphorus metabolism studies in such cases.

We have never seen any findings in patients with either form of arthritis that would suggest the existence of hyperparathyroidism as a causal factor. True, many of the cases of rheumatoid arthritis show x-ray evidence of decalcification (bone atrophy). However, this is no more than we often see associated with disuse. For instance, we know that the calcium excretion of a patient when immobilized in a cast is much higher than when this same patient is allowed ordinary ward activity[21]. To date, we have studied the total calcium and phosphorus metabolism of a series of cases, some of them having rheumatoid arthritis and the others having osteoarthritis (hypertrophic)[21]. We took particular pains to see that we had chosen cases of rheumatoid or proliferative arthritis that showed marked bone atrophy and cases of degenerative or hypertrophic arthritis showing marked proliferative bone changes. As a result of these studies we can state that we found no changes in the serum calcium or serum phosphorus or the calcium and phosphorus metabolism of a degree sufficient to enable one to make a diagnosis of hyperparathyroidism. The alterations in the calcium and phosphorus metabolism observed were hardly any more marked than one obtains in a study of a large series of normal individuals observed on the same régime. Another reason why we are skeptical about arthritis occurring as a result of hyperparathyroidism is the fact that in the past five years at the Massachusetts General Hospital we have never seen the two diseases in the same patient, although we have had the privilege of seeing seven cases of hyperparathyroidism. All seven cases satisfied all the requirements necessary to make such a diagnosis and yet no one of them had arthritis. One patient has been known to have the disease for fifteen years[20].

There are other factors which are mentioned as playing a rôle in the production of arthritis such as gastro-intestinal disturbances, constipation, high carbohydrate diet, carbohydrate indigestion, endocrine disorders, metabolism disorders, etc. Our proof that such factors are operative in the production or the continuation of the disease is anything but complete. We must ask ourselves how are they operative? Can they produce the joint changes we observe? Does remedying them cure the disease or are the cures reported nothing more than natural remissions?

Therefore, in summarizing, one can say that rheumatoid arthritis is a disease of unknown etiology, characterized by remissions and relapses. Natural remissions may very easily be interpreted as cures, therefore the need of a definition of the word, cure, when used in connection with this disease. We have presented certain data concerning the anatomy and physiology of normal joints, the origin and nature of normal synovial fluid, the pathology and physiology of joint effusions as well as to point out how this knowledge might better enable us to understand the manner in which the intra-articular changes might be produced. While it is true that some of the statements we have set forth are not fully proved, they do suggest methods of approach which may lead us to a better understanding of this disease.

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TUMORS OF THE SMALL INTESTINE*

With a Few Remarks Concerning the Surgical Treatment of Small Intestinal Tumors by Dr. Frank H. Lahey

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THE rarity of small intestinal tumors is well recognized. Of the tumors originating in the intestinal tract between the pylorus and the ileocecal valve, carcinoma is the most common, and benign adenoma is second in frequency of occurrence. Sarcomas, lipomas, chronic inflammatory tumors, fibromas, myomas and carcinoid tumors occur in the relative frequency of the order given. Lymphangiomas, hemangiomas, pancreatic rests, hematomas and endotheliomas are extremely rare, but make up the list of tumors which are found to be primary in this region.

Statistics of large series of postmortem examinations show cancer in about 10 per cent of bodies. According to Ewing[1] 8.56 per cent of all cancers are primary in the intestinal tract exclusive of the stomach. He states that carcinoma of the duodenum accounts for only four per cent and carcinoma of the jejunum and ileum only three per cent of the intestinal cancers. Ninety-three per cent of intestinal carcinomas are primary in the colon and rectum.

Because of the extreme infrequency with which cases of small intestinal tumors occur, the several hundred cases which are now in the literature have been reported for the most part in groups of one, two and three cases. The task of studying the condition, however, has been very much lightened by several able reviews of the literature which have been published in recent years. Speese[2] collected and reviewed the cases of sarcoma of the small intestine in 1914. Ullman and Abeshouse[3] in 1932, studied lymphosarcoma of the small and large intestine. Clark[4] and others have covered the literature on carcinoma; Cooke[5] has reported the various pathologic aspects of carcinoid tumors; Milles[6] has discussed the rare condition known as lymphangioma of the intestine, and Cattell[7] has reported from this Clinic a case of leiomyosarcoma. It is rare for one individual to see more than eight or ten cases of small intestinal tumors during a lifetime of medical or surgical practice. Reports on series of considerable numbers of cases have come only from the large clinics and hospitals. Rankin and Mayo[8] reported on the cases of carcinoma of the small intestine seen at the Mayo Clinic up to 1930. Raiford[9] has made a comprehensive study of all types of tumors found in the small intestine of patients who have been treated at the Johns Hopkins Hospital.

Raiford found in his study of eighty-two cases that 40 per cent of the tumors were malignant and 60 per cent were of the benign type. Thirty-seven per cent of the patients come to operation because of symptoms produced by the tumor. However, 46 per cent of the tumors were asymptomatic and were found incidentally at autopsy.

Tumors of the small intestine may be classified according to their gross characteristics into annular tumors and asymmetric tumors. Annular tumors are usually malignant and are usually carcinomas. Sarcomas sometimes form an annular lesion, but their tendency is toward the expansive type of growth. The majority of tumors are of the asymmetric type which may be further subdivided into internal and external types. The internal tumors are either pedunculated or attached to the wall by a broad base and grow inward toward the lumen of the intestine. Benign tumors are frequently of this type. The external type is most apt to be malignant and their growth is by extension outward either into the peritoneal cavity or into the mesentery. This is the type that produces the large tumors which sometimes occur in the small intestine.

The symptomatology of cases of small intestinal tumor is variable and seldom conforms to any definite clinical picture. The mechanical condition of the bowel which is dependent upon the size and location of the growth is the most important factor in the determination of the nature of the symptoms produced. Invasion of other viscera, and cachexia eventually enter into the clinical picture. Sarcoma is more productive of constitutional symptoms than is carcinoma. Malignant tumors and some benign growths tend to bleed into the intestines, resulting in a secondary anemia.

The duodenum is the most common site of carcinoma of the small bowel if cancer of the ampulla of Vater is included. The ileum is next in frequency of location with the jejunum being the least common site of carcinoma. Sarcoma, however, as well as benign adenoma is much more frequently found in the ileum than elsewhere in the small intestine or colon. This is probably due to the greater abundance of lymphoid tissue in this region.

Carcinoma of the first portion of the duodenum has been known to follow an ulcer in this

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region in very rare instances. Even in the cases reported the relationship between the ulcer and the later malignancy has lacked convincing proof. The symptoms of cancer in this region are the same as for pyloric obstruction from any other cause. Carcinoma at or near the ampulla of Vater is characterized by complete and uninterrupted jaundice of the obstructive type, dilatation of the gall bladder and a short, rapidly fatal course. It cannot be differentiated clinically from carcinoma of the head of the pancreas. Obstruction of the duodenum distal to the ampulla may result in the vomiting of much bile. The obstruction in the duodenum and upper jejunum is slowly progressive and results in severe cramplike epigastric pain which tends to be at its greatest intensity two or three hours after meals. Vomiting is an early symptom and increases in frequency and in amount as the obstruction increases. It is well known that complete obstruction of the upper intestinal tract produces vomiting of large amounts of gastric, biliary and pancreatic secretions. This in turn causes rapidly progressive dehydration, toxemia, alkalosis and prostration. In obstruction of the duodenum the sensation of fullness and distention may become very distressing to the patient.

In the jejuno-ileal region the most common evidence of tumor is obstruction which is either slowly progressive and recurrent or acute in type. In the gradual encroachment upon the lumen either by a malignant growth encircling the intestine or by an internal tumor protruding into the lumen, the patient frequently goes along for several months with symptoms which are not marked but are fairly constant and progressive. Pain is diffuse, shifting in character and located either in the lower or upper abdomen; it may be dull and aching or sharp and cramp-like. Nausea and vomiting are usually present but are not marked; the patient vomits once or twice a week or even less frequently. Malaise, anorexia and loss of weight are usual complaints. These general symptoms are more marked in cases with malignant tumors. The gradual and progressive encroachment upon the lumen of the intestine gives rise to intermittent attacks of intestinal obstruction with abdominal pain, vomiting, dehydration and prostration. The attack may subside after a few hours or a day only to recur at steadily decreasing intervals. Although it is seldom that the lumen of the intestine becomes completely occluded, intermittent or permanent obstruction may be produced by food particles which do not pass readily by the portion narrowed by the growth. Although the small intestine is notoriously intolerant to acute obstruction and distention, it possesses a real capacity for adaptation to slow progressive obstruction. The gut may be markedly dilated above the point of obstruction but the musculature of the wall undergoes extensive hypertrophy so that the wall may be one half inch in thickness and is therefore able to supply the force necessary to overcome the obstruction.

Instead of a gradual onset, symptoms may begin with dramatic suddenness. Intussusception, which occurs in about 30 per cent of tumors of the jejunum and ileum, is manifested by a sudden, sharp, agonizing pain which does not subside for several hours. The explanation of this common complication is assumed to be the gripping of the tumor by a peristaltic wave which telescopes a section of the intestine into the section below. Spontaneous reduction is much less common in these cases than it is in the acute intussusception of children. If it does occur the pain is immediately relieved. Vomiting starts immediately after the onset of pain and is usually continuous and severe. Shock does not occur immediately but develops within an hour or so. Distention, particularly in high level obstructions, may not be marked and may even be absent. After a time tenesmus with a discharge of bloody mucus from the rectum occurs. Usually an abdominal mass can be palpated. Characteristically it is sausage-shaped and is hard and rigid during the attacks of pain. It may change in size and position from day to day. Other physical signs in a patient with an acute intussusception are secondary to the state of shock. The characteristic cold, clammy skin, the rapid thready pulse, the rapid shallow respiration and the subnormal temperature make up the clinical picture.

In the case that is not acutely obstructed the physical findings are mainly those relative to cachexia of more or less degree combined with abdominal findings which may or may not be present. In long-standing partial obstruction there is usually some distention but it may not be marked. Active peristalsis may be both visible and audible. An abdominal mass is usually palpable. It may be freely movable but is often fixed.

In a small group of cases the tumor grows away from the lumen of the bowel and therefore does not produce mechanical obstruction. In these cases the clinical picture is obscure because the symptoms are dependent upon pressure and the constitutional effect of malignant disease. The physical examination shows a secondary anemia but is essentially negative until the growth becomes large enough to be palpable.

Among the complications which may occur is acute perforation of the intestine at or above the point of obstruction, as happened in one of our own cases. Gross hemorrhage is very rare.

The roentgenologic examination often fails to

locate the trouble, due chiefly to the difficulty in examining the small intestine as compared to the rest of the gastro-intestinal tract. The best method of detecting small intestinal lesions is by film examination in a series of films taken every hour for five hours following an opaque meal. Dilatation of the small intestine with delay in the passage of barium is the most frequent positive x-ray sign. A considerable length of intestine may be dilated and show as irregular distended coils.

Figure 1, shows quite well the extensive dilatation of the small intestine which occurred in a man (Case No. 1) with almost complete obstruction due to carcinoma of the lower jejunum. At operation these coils were found to be dilated to a lumen four inches in diameter.

Sometimes the barium column appears to be blocked off or narrowed at a definite point, as shown in figure 2, of a woman (Case No. 5) with a carcinoma of the jejunum near the ligament of Treitz.

Filling defects and craters are very seldom demonstrated in the small intestine. Occasionally in carcinoma of the ampulla of Vater a crater-like filling of the ampulla may be seen.

Although at some point in the progress of the disease definite x-ray signs usually develop, it is often after the lesion becomes inoperable that an x-ray diagnosis can be made. It is important to bear in mind that a negative gastro-intestinal series does not rule out malignancy of the small bowel.

Clinical laboratory examination shows a secondary anemia in all malignant cases and in many benign cases if allowance is made for the concentration of the blood from dehydration in patients with vomiting.

The examination of the stools for occult blood while the patient is on a meat-free diet is probably the most important clinical test. The value of this examination is stressed by nearly all authors discussing the diagnosis of small intestinal tumors.

Gastric achlorhydria is a frequent finding.

In cases with considerable vomiting, chemical blood studies show diminished chlorides, a high CO_2 content and a high non-protein nitrogen.

The urine shows evidences of dehydration and of acute toxic nephritis. Complete suppression of urine is not an uncommon postoperative complication.

The diagnosis of tumor of the small intestine is not often made before operation or postmortem. The very rarity of the condition discourages the diagnosis. However, if the condition is kept in mind, the x-ray and physical examinations carefully done and the stools are examined for occult blood, the diagnosis at least of a small intestinal lesion can sometimes be made. A diagnosis as to the type of lesion is usually impossible. Sarcoma is more apt to occur in younger people; the first, third and fourth decades embrace most of the incidence of sarcoma. Obstruction due to constricting adhesions, obstruction due to volvulus or strangulation, spontaneous intussusception, appendicitis and tumors elsewhere in the gastro-intestinal tract are among the conditions to be differentiated in the diagnosis.

The treatment of tumors of the small intestine is entirely surgical. Patients who are acutely obstructed and in poor condition should have only an enterostomy at the first operation. Later resection of the tumor and an end to end or side to side anastomosis may be done after the patient's condition has been improved by replenishing the body fluid and salts with repeated or continuous intravenous injections of saline and glucose solutions. In cases where the growth has extended and metastasized, a side tracking lateral anastomosis around the obstruction may give relief for the remainder of life.

The prognosis in malignant disease of the small intestine is usually poor. Carcinoma of the small intestine tends to metastasize early to the mesenteric glands. Lymphosarcomas extend rapidly to surrounding tissues. The spindle cell sarcoma is uncommon but is more apt to remain localized. Rankin and Mayo reported no case living longer than three years. The average was less than one year. Cases have been reported that have lived as long as ten years after operation without a recurrence. Of our series there is one case of carcinoma that is still alive and well nearly five years after his operation for the removal of a carcinoma of the jejunum.

The series reported here consists of thirteen cases. Cases 3 and 4 have been published before by Dr. Lahey[10] in 1915. Case No. 10 was reported by Dr. Cattell[7] in 1931. Eleven of the thirteen tumors were carcinomas of which three were located at the ampulla of Vater, six were in the jejunum and two were found in the ileum. There was one instance of leiomyosarcoma of the jejunum and one of lymphangioma of the ileum. The following are abstracts from the case records of these patients. It is to be noted that in the three cases of carcinoma of the ampulla of Vater the diagnosis was not proved by histologic study of tissue, but the histories are so typical that it seems worth while to include them in this report.

CASE No. 1.

Mr. L. B. (Clinic No. 2797), aged 43, was referred to the Clinic by Dr. Parker Mills of Providence, R. I., on January 18, 1928. For a period of one and one-half years the patient had had distention, generalized diffuse abdominal pain which was not severe, borborygmus and flatus. His appetite had been good but he had lost ten pounds in weight. The bowels had moved two or three times daily. No blood or mucus was noted in the stools. There was no vomiting.

Gastro-intestinal x-rays had been taken elsewhere

in September, 1927, without disclosing any cause for the symptoms.

Physical examination was not remarkable save for the abdomen which showed visible and audible peristalsis and some distention. By rectum a hard nodule could be felt through the anterior wall of the rectum.

A plain x-ray film of the abdomen showed the jejunum to be tremendously distended and lifted high up in the abdomen. A barium meal showed the stomach crowded up high under the diaphragm. In the afternoon nearly all of the barium was in the dilated jejunum, there being only a trace found in

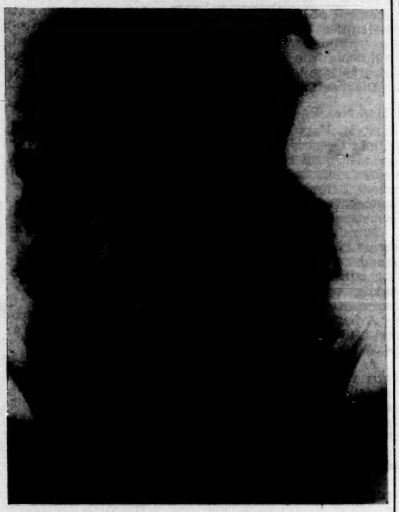

FIGURE 1. This roentgenogram of Case No. 1 shows the enormous dilatation of the small intestine which developed from chronic progressive obstruction of the lower jejunum. Lines have been drawn on the film to indicate the outlines of the distended loop.

the cecum. On the following morning there was only a small amount of barium that had passed through into the colon. The blocking appeared to be in the pelvic area somewhere in relation to the fifth lumbar vertebra.

The preoperative diagnosis was chronic obstruction of the small intestine.

At operation (Dr. Clute) several small hard calcified glands were found in the pelvis along the sigmoid.

The upper fifteen feet of small intestine was found to be huge, dilated and hypertrophied. It was not distended but measured approximately four inches in diameter. The intestinal wall was nearly one centimeter in thickness due to hypertrophy. At approximately fifteen feet from the ligament of Treitz there was almost complete obstruction by a hard, indurated, nodular mass about the size of two thumbs, causing a scar drawing the entire mass over to the mesentery border. There were several enlarged hard glands in the mesentery at this position. One small gland was removed for section and an enterostomy was done eight inches above the obstruction.

Pathologic examination of the gland revealed metastatic carcinoma.

At a second operation ten days after the first one, the section of jejunum containing the mass was resected along with the glands in the mesentery.

Pathologic examination showed carcinoma simplex of the jejunum.

The postoperative recovery was satisfactory and the patient was living and well in October, 1932, nearly five years after his operation.

Diagnosis: Carcinoma of the jejunum.

CASE No. 2.

Mr. H. P. C. (Clinic No. 29727), aged 61, was referred to the Clinic by Dr. James Quinn of Springfield, Massachusetts. He had always been well until six weeks before admission. At that time he started to vomit about three hours after meals. He had no definite pain, but complained of distress and fullness in the epigastrium. The vomitus was liquid in character with some food particles. The vomiting had increased in severity until the patient was vomiting one or more times daily. The weight loss amounted to forty pounds. The bowel function had been normal except for some diarrhea two weeks before admission.

The physical examination showed him to be well nourished but there was evidence of recent weight loss. The remainder of the examination was irrelevant.

Laboratory examinations showed the urine to be negative except for a very slight trace of albumin. The non-protein nitrogen was elevated to 114 mg. per 100 cc. of blood. The red blood count was 4,300,000. The hemoglobin was 80 per cent. The gastric analysis showed a free hydrochloric acid of 29 and a total acidity of 42. The stools were consistently positive for occult blood.

X-ray examinations by means of a barium meal showed a normal stomach. Serial films taken at hourly intervals showed stasis in a loop of jejunum which was considerably dilated. After twenty-four hours there was still barium in this loop.

A diagnosis of carcinoma of the small intestine was made.

On a liquid diet all signs and symptoms of obstruction disappeared. The non-protein nitrogen fell to normal.

At operation (Dr. Clute) an annular carcinoma of the jejunum was found and resected. There were no evident metastases. A side to side anastomosis and a jejunostomy was performed.

The patient recovered and left the hospital in good condition.

CASE No. 3.

Mr. A. L., aged 45, was seen January 17, 1914, complaining of frequent vomiting, weakness, pallor and constipation. He had begun to have symptoms six months previously. Weight loss amounted to twelve pounds. There had been no melena.

Physical examination showed him well-developed and nourished, but cachectic in appearance. The hemoglobin was 70 per cent (Tallqvist). There was considerable tenderness in the right upper quadrant of the abdomen and on deep palpation an irregular, hard mass extending downward was felt.

At operation (1914, by Dr. Lahey) a small annular mass completely surrounding the intestine was found in the jejunum about ten inches from the ligament of Treitz. The lumen which remained was just large enough to admit the finger tip. There were few enlarged glands in the mesentery which were not removed. Resection of the mass with about eighteen inches of intestine was done and an end to end anastomosis was accomplished.

Pathologic diagnosis was carcinoma of the jejunum.

The postoperative course was satisfactory, but in the following months after leaving the hospital the patient gradually failed. An exploratory laparotomy

done six months later, June 22, 1915, showed extensive carcinoma throughout the abdomen.

Diagnosis: Carcinoma of the jejunum.

CASE No. 4.

Miss L. C., aged 60, was seen on October 4, 1913, for dyspeptic symptoms of four months' duration. For the past month she had been constipated and had colicky abdominal pain particularly in the left lower quadrant. She had vomited only once. There was no melena. The weight loss amounted to thirty pounds.

Physical examination showed her to be pale and poorly nourished. Otherwise the examination was negative.

At operation (Dr. Lahey) a small discrete mass was found in the small intestine several feet from the ileocecal valve. Resection of the mass with about twelve inches of intestine was done and an end to end anastomosis was accomplished.

The pathologic diagnosis was carcinoma of the small intestine.

The postoperative recovery was uneventful but the patient died of probable recurrence one year later.

Diagnosis: Carcinoma of the ileum.

CASE No. 5.

Mrs. A. J. (Clinic No. 7128), aged 69, came to the Clinic November 7, 1928, with the chief complaint

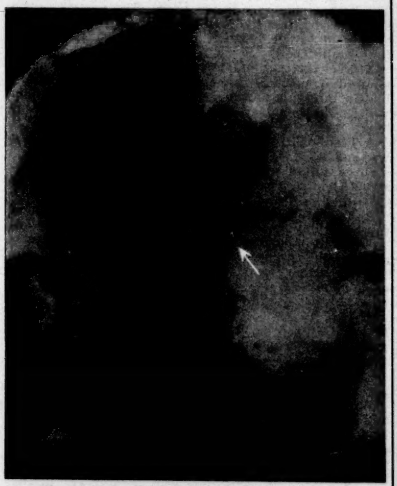

FIGURE 2. In this roentgenogram of Case No. 5 the arrow points to an irregular filling defect near the juncture of the duodenum and the jejunum. There is partial obstruction to the passage of the barium and marked dilatation of the proximal portion of the duodenum.

of vomiting. The onset of her illness was about four months previously with attacks of epigastric discomfort and vomiting. For the two months preceding admission she had been able to retain only liquids and frequently vomited large quantities. There was no melena. The weight loss was fifteen pounds. Gastro-intestinal x-rays in August at another Clinic had shown a six hour residue. Another examination in October, before admission to our clinic, was essentially negative.

The physical examination showed the blood pressure to be 170 mm. systolic and 90 mm. diastolic. The heart was moderately enlarged. The pupils were unequal. The Wassermann was negative.

Roentgen examination of the gastro-intestinal tract by the gastro-enterological service in the clinic showed marked dilatation and retention of barium in the third portion of the duodenum and the first portion of the jejunum and a diagnosis of a lesion of the jejunum was made.

At operation (Dr. Lahey) an annular, constricting mass was found in the jejunum very near to the ligament of Treitz. It was so fixed that the technical difficulties appeared to preclude removal. A gastro-enterostomy was done. The patient left the hospital following the operation, but death occurred three months later in February, 1929.

Diagnosis: Carcinoma of the jejunum.

CASE No. 6.

Mrs. M. F., (Clinic No. 18811) aged 61, came to the Clinic October 21, 1930. The chief complaints were an afternoon rise of temperature and fatigue of two years' duration. She had had rheumatic fever nine years before. She had taken iron many years for anemia. She had always had to take cathartics for constipation.

Physical examination showed pallor, rheumatic heart disease and a firm fixed mass in the right lower quadrant of the abdomen. The red blood count was 3.2 million; the hemoglobin 50 per cent; the gastric analysis showed a free Hcl of 30; and the stools were positive for occult blood.

A barium series showed no definite pathology except that in six hours after the meal there was a coil of jejunum containing barium.

At operation (Dr. Clute) a mass the size of a fist was found to involve the jejunum and was situated eight inches below the ligament of Treitz. It was adherent to the appendix and to the posterior parietal peritoneum. There were few enlarged glands in the mesentery. The entire involvement was removed, including ten inches of jejunum and an end to end anastomosis was done.

The pathologic diagnosis was adenocarcinoma, grade 2, of the jejunum with metastasis to the lymph nodes.

After the operation the patient passed very little urine, the nonprotein nitrogen rose and she died in coma on the second postoperative day.

Diagnosis: Carcinoma of the jejunum.

CASE No. 7.

Mrs. K. T. (Clinic No. 26789), aged 46, came to the Clinic in March, 1932, complaining of increasing constipation, loss of weight and abdominal pain. Two years before she had been treated for four months for epigastric pain and was relieved. She felt well for a year and regained weight. Five months before coming to the Clinic the pain recurred with flatulence, excessive belching and abdominal borborygmus. The abdomen became very tender and sensitive. She occasionally vomited sour material. The pain was always worse after vomiting and was made worse by food. With the pain she noted "lumps" over the abdomen and obtained relief by massaging the "lumps" until they disappeared. There was a loss of twenty-nine pounds in weight during the five months. Constipation had become progressively more severe. Enemata or laxatives were required for bowel action.

Physical examination showed her to be poorly nourished. The abdomen was flat. The physical examination was otherwise not remarkable.

The red blood count was 4.5 million; hemoglobin 80 per cent (Tallqvist); and the white count was 6,650. The gastric analysis showed a free Hcl of 53.

The urine was normal. X-ray examination showed positive shadows of gall stones. A barium series failed to reveal any evidence of other pathology.

At operation (Dr. Lahey) a constricting tumor of the jejunum was found about twelve inches below the ligament of Treitz. The jejunum above was slightly dilated and the wall was thick and hypertrophied. The spleen was slightly enlarged. In the mesentery of the involved jejunum were several enlarged lymph nodes, none of which were firm. The tumor was resected along with about six inches of intestine on either side of the mass and an end to end anastomosis was done.

The pathologic diagnosis was malignant adenoma of the jejunum.

The postoperative course was satisfactory until the seventh day when she developed intestinal obstruction from intussusception of the jejunum into the ileum. She was operated upon again but death occurred on the thirteenth day after the first operation.

Diagnosis: Carcinoma of the jejunum.

CASE No. 8.

Miss N. W., aged 62, was referred to the Clinic on November 6, 1926, by Dr. E. T. Mannix of Lynn, Massachusetts. For two months the patient had complained of gaseous eructations, anorexia, loss of twelve pounds in weight, constipation and some vomiting. She had recently noted that she was pale.

The essential physical findings were moderate pallor and a large mass in the pelvis extending above the crests of the ileum.

Roentgenologic examination by means of a barium meal showed four hours after the meal was given a dilated loop of small intestine. This loop was still dilated and contained barium after twenty-four hours. A barium enema showed no involvement of the colon.

At operation (Dr. Lahey) a mass the size of a large orange was found in the mid portion of the ileum. There were few enlarged glands in the mesentery. The mass with about eighteen inches of intestine, including the mesentery was resected and an end to end anastomosis was done.

Pathologic examination showed the tumor to be an adenocarcinoma of the ileum, grade 3, malignancy.

Death occurred five days after operation of intestinal obstruction and peritonitis.

Diagnosis: Carcinoma of the ileum.

CASE No. 9.

Mr. L. H., (Clinic No. 19648) aged 42, came to the Clinic March 21, 1927, with an ileal fistula which had followed an operation elsewhere two months before for an acutely strangulated hernia of small intestine through a congenital defect at the base of the ileal mesentery. His postoperative course had not been unusual but the final closure had not yet been done.

Physical examination was not remarkable.

No x-ray gastro-intestinal studies were done. Roentgen examination of the chest was essentially negative.

At operation (Dr. Lahey) the intestine appeared to be involved in an extensive tuberculous process. There were multiple tubercles over many areas.

Operative diagnosis: Tuberculous peritonitis.

Pathologic diagnosis: Lymphangioma; chronic inflammation.

Living and fairly well in December, 1930.

Diagnosis: Lymphangioma of the ileum.

CASE No. 10.

Mr. J. C., (Clinic No. 18275) aged 40, came to Clinic September 16, 1930, several hours after the sudden onset of severe cramplike abdominal pains. He had vomited only once and the bowels had moved with enemas.

On examination he appeared to be in shock. The abdomen was rounded and prominent. No mass was felt. No free fluid was demonstrated. The white blood count was 12,200.

At operation (Dr. Cattell) which was done five hours after the onset of pain, the upper abdomen contained considerable turbid fluid, evidently intestinal content. A pedunculated growth eight or nine centimeters in diameter with a cauliflowerlike surface was found attached to the upper part of the jejunum. There was a perforation directly through the tumor into the abdominal cavity. The tumor was resected; end to end anastomosis was made.

Death occurred on the fifth day postoperatively of acute progressive uremia. In spite of intravenous saline and glucose solutions the patient died in coma with a nonprotein nitrogen of 240 mg. per 100 cc. blood.

Pathologic examination of the tumor showed it to be a leiomyosarcoma of the jejunum.

Diagnosis: Leiomyosarcoma of the jejunum.

CASE No. 11.

Mrs. M. G. (Clinic No. 9491), aged 60, was referred to the Clinic on April 16, 1929, by Dr. E. P. Hodgdon, for pruritus and jaundice. The pruritus, which was very severe, began three months before admission and jaundice was first noticed a month later. Except for one day she had had no pain, but had experienced a constant discomfort in the right upper quadrant. At the onset she had one attack of pain accompanied by vomiting. The bowels were constipated; the stools were clay colored and the weight loss was twenty pounds.

The physical examination showed her to be deeply jaundiced. The gall bladder was enlarged and palpable.

Gastro-intestinal x-rays showed no evidence of disease of the stomach and the intestines.

At operation (Dr. Clute) the gall bladder was found to be distended. There was no evidence of biliary cirrhosis. There was thickening of the head of the pancreas at the region of the ampulla. A cholecystogastrostomy was done and the patient made a good postoperative recovery.

Presumptive diagnosis: Carcinoma of the ampulla of Vater.

CASE No. 12.

Mr. G. H. N. (Clinic No. 2844), aged 70, came to the Clinic on January 23, 1928, with the complaint of painless, persistent jaundice of three weeks' duration. He had gradually lost weight for a period of three years. Two weeks before admission he had great difficulty in getting the bowels to move, but his bowel function was normal on admission. The stools were clay colored.

Physical examination showed marked jaundice. There was evidence of recent weight loss. The liver was moderately enlarged and the gall bladder was distended and palpable. The heart was enlarged. A loud systolic murmur could be heard over the fourth left interspace. Auricular fibrillation was present.

The urine contained a few casts and a very slight trace of albumin. The serum bilirubin was 12.5 mg. per 100 cc. The non protein nitrogen was 43 mg. per 100 cc. of blood. The blood Wassermann was negative.

X-ray examination of the stomach by means of a barium meal showed some irregularity of the antrum. The duodenum appeared to be normal.

At operation (Dr. Clute) considerable free fluid was found in the abdomen. There was marked enlargement of the gall bladder. The liver showed extensive hepatitis with a gray, granular surface. The stomach was normal. A nodule was felt in the pancreas at the entrance of the common duct into the duodenum. A cholecysteneterostomy was done.

The patient gradually weakened and died twenty-three days after the operation. There was no autopsy.

Presumptive diagnosis: Carcinoma of the ampulla of Vater.

CASE No. 13.

Mr. J. F. (Clinic No. 24532) came to the Clinic October 2, 1931, complaining of attacks of fever and jaundice. He had had vague cramp-like abdominal pains for eighteen years. In 1926, he had gastro-intestinal x-rays elsewhere which were negative. In 1928, he had epigastric pain that came on two or three hours after meals and was relieved by food or by soda. He was x-rayed again and a diagnosis of duodenal ulcer was made. He was relieved by a modified Sippy diet and he felt well for one year. He then began to have attacks of jaundice and moderate fever lasting a few days. The skin was very itchy. The jaundice was not severe, but never fully disappeared. The urine had shown a trace of sugar at times. He had lost forty-five pounds in three years.

Physical examination showed marked jaundice and loss of weight. The liver was felt two fingerbreadths below the costal margin. There were no other masses palpable. There was no tenderness or rigidity.

Laboratory tests showed a trace of sugar, a very slight trace of albumin and a strong test for bile in the urine. The serum bilirubin was 2.4 mg. per 100 cc. diphasic. The blood sugar was 0.08 per cent. The NPN was 28 mg. per 100 cc. of blood. The RBC was 4.8 million; WBC 11,500; hemoglobin 75 per cent. The stools contained bile but no occult blood. The urine urobilinogen was positive in 1-100 dilution. The galactose tolerance test showed an excretion of 1.5 grams in five hours after the ingestion of 40 grams of galactose.

X-ray examination by means of a barium meal, showed a normal, hyperactive stomach. The second portion of the duodenum appeared dilated and fixed.

At operation (Dr. Lahey) the gall bladder was distended. The walls were thickened. There were no stones. The common duct was distended. At the location of the ampulla of Vater was felt a hard indurated lesion. A cholecystjejunostomy was done.

The patient made a good postoperative recovery and the jaundice cleared up.

In February, 1932, he developed complete obstruction of the biliary tract as evidenced by a return of jaundice and absence of bile in the stools.

When last seen in September, 1932, his condition was very poor. He had ascites, a palpable mass in the right upper quadrant and x-ray evidence of metastases in the lungs.

Probable diagnosis: Carcinoma of the ampulla of Vater.

NOTE BY DR. FRANK H. LAHEY

The indications for surgical treatment of tumors of the small intestine are so definite that but very little needs to be said on the subject.

From Dr. Kiefer's discussion it is obvious that the most difficult part of the problem is to recognize this lesion early enough for the prognosis to be hopeful from a surgical point of view.

The relative rarity of small intestine tumors makes it impossible for anyone to have had a large experience with this lesion. As the result, however, of our experience, there are one or two points in the surgical management of these cases which may be of value, the most important one being, I think, the advantage of preliminary lateral anastomosis around the tumor and secondary removal after the fecal stream has been established through the new lateral enterostomy and intestinal obstruction has been overcome. Everyone who is familiar and experienced with the technical side of surgery realizes that no matter how carefully end to end anastomoses of small intestine are done, there tends to be some degree of mechanical obstruction of a temporary character. When this is superimposed upon an obstruction which has already existed for some days, it will in many cases result in a fatality which could very probably have been avoided had a preliminary lateral anastomosis been done.

Another technical feature from the surgical point of view, which is of value in these cases dealing with this same aspect of the problem, is the addition of jejunostomy above the point of anastomosis in those cases where primary resection and anastomosis, either end to end or lateral, is deemed advisable.

As a result of our own experience with the surgery of this lesion, the factor which is most likely to bring about a fatality is obstruction, with its consequent dehydration and intoxication, rather than the danger of leakage. Even in those early lesions where one is tempted to do immediate resection and anastomosis, the ultimate mortality will be, we believe, lower with preliminary lateral anastomosis and secondary removal of the lesion at a later date.

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A BRIEF SUMMARY OF RESULTS IN THE TREATMENT OF CARCINOMA OF THE ENDOMETRIUM*

BY GEORGE V. SMITH, M.D.‡

THE material for this report comprises 181 cases of adenocarcinoma of the endometrium treated before September, 1929, by surgery or radium or a combination of surgery and radium. This series includes only those cases in which the diagnosis was made or confirmed by microscopic examination†. Sarcoma of the endometrium, as well as carcinoma, was found three times. One case had also early squamous carcinoma of the cervix. Recurrent carcinoma of the breast was present in another case. In one instance sarcomatous change in a fibroid was diagnosed. Three cases, so far as could be determined, had also primary cancer of the ovary.

The youngest patient was 36 years old at the time of treatment; the oldest, 85. Eighty per cent of the group were over 50 years of age. Forty-six per cent had never been pregnant; 2.7 per cent had had only abortions or miscarriages; the remainder had had from one to nine children. Eighty-one per cent had passed the menopause.

GROSS RESULTS

TOTAL NUMBER OF CASES TREATED BEFORE SEPTEMBER, 1922 — 85

Operative deaths:
- Good prognosis — 5

Untraceable since the time of operation:
- Good prognosis — 1
- Guarded prognosis — 1
- Total — 2

Died of recurrence less than 3 years after operation:
- Good prognosis — 4
- Guarded prognosis — 7
- Poor prognosis — 16
- Total — 27

Died of cerebral hemorrhage 3 months after operation:
- Poor prognosis — 1

Died of recurrence 3 to 5 years after operation:
- Guarded prognosis — 1
- Poor prognosis — 1
- Total — 2

Died of angina pectoris 3 years, 4 months after operation:
- Good prognosis — 1

Died of recurrence 5 to 8 years after operation:
- Good prognosis — 5
- Guarded prognosis — 3
- Poor prognosis — 1
- Total — 9

Well 6½ to 9 years after operation—untraceable since:
- Good prognosis — 4

Died of other causes 7 to 8 years after operation:
- Good prognosis — 5
  - 1—"Angina pectoris"
  - 2—"Arteriosclerosis"
  - 3—"Heart disease"
  - 4—"Acute obstruction; operation; pulmonary embolus"
  - 5—"Nephritis"

Well 10 to 21 years after operation:
- Good prognosis — 22
- Guarded prognosis — 2
- Poor prognosis — 2
- Total — 26

Died of other causes 10 to 12 years after operation:
- Guarded prognosis — 3
  - 1—"Arteriosclerosis"
  - 2 and 3—"Cerebral hemorrhage"

Total — 85

Alive 3 years after operation—50 (62.5 per cent of the 80 who survived operation)

Alive 5 years after operation—47 (58.7 per cent of the 80 who survived operation)

Alive 10 years after operation—29 (36.2 per cent of the 80 who survived operation)

TOTAL NUMBER OF CASES TREATED FROM SEPTEMBER, 1922, TO SEPTEMBER, 1927 — 67

Operative deaths:
- Good prognosis — 2

Untraceable since the time of operation:
- Good prognosis — 2

Died of bronchopneumonia 6 months after operation:
- Good prognosis — 1

Well 1 year after operation—untraceable since:
- Good prognosis — 1

Well 2 years, 5 months, after operation—untraceable since:
- Good prognosis — 1

Died of recurrence less than 3 years after operation:
- Good prognosis — 3
- Guarded prognosis — 3
- Poor prognosis — 14
- Total — 20

Died of cerebral hemorrhage 3 to 4 years after operation:
- Good prognosis — 3

Died of recurrence 3 to 5 years after operation:
- Good prognosis — 1
- Guarded prognosis — 4
- Poor prognosis — 1
- Total — 6

Died of recurrence 5 to 6 years after operation:
- Good prognosis — 1
- Guarded prognosis — 3
- Total — 4

Well 5 to 10 years after operation:
- Good prognosis — 20
- Guarded prognosis — 4
- Poor prognosis — 3
- Total — 27

Total — 67

*From the Free Hospital for Women, Brookline, Mass.

†Drs. S. B. Wolbach and S. Warren very kindly checked the diagnosis in 61.5 per cent of the cases which were alive five years after treatment.

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Alive 3 years after operation—40 (61.5 per cent of the 65 who survived operation)

Alive 5 years after operation—31 (47.6 per cent of the 65 who survived operation)

TOTAL NUMBER OF CASES TREATED FROM SEPTEMBER, 1927, TO SEPTEMBER, 1929..... 29

Operative deaths:
Good prognosis..... 2

Died of agranulocytic angina 2 years, 2 months after operation:
Good prognosis..... 1

Died of recurrence less than 3 years after operation:
Good prognosis..... 4
Poor prognosis..... 7 } 11

Alive with recurrence 3 years after operation:
Good prognosis..... 1

Well 3 to 5 years after operation:
Good prognosis..... 13
Poor prognosis..... 1 } 14

Alive 3 years after operation—15 (55.5 per cent of the 27 who survived operation)

Thus: Of the 85 cases treated before September, 1922, 34.1 per cent were alive 10 years after operation.

Of the 152 cases treated before September, 1927, 51.3 per cent were alive 5 years after operation.

Of the 181 cases treated before September, 1929, 57.4 per cent were alive 3 years after operation.

Those patients showing no gross or microscopic evidence of extension or metastasis of the disease at the time of operation were given a good prognosis. A guarded prognosis was assigned to those treated by radium alone in whom it was impossible to be certain whether the disease had reached beyond the uterine cavity, and to a few others in whom the disease at operation was found to have extended to the serosa of the uterus. Patients with obvious extension through the serosa or into the parametria and with vaginal, pelvic or omental metastases were given a poor prognosis. The following table shows the results when prognosis is considered:

To September, 1929—Total..... 181
Good prognosis..... 103
Poor or guarded prognosis 78

Alive 3 years after operation:
Good prognosis..... 70 (67.9 per cent)
Poor or guarded prognosis ..... 27 (34.6 per cent)

To September, 1927—Total..... 152
Good prognosis..... 82
Poor or guarded prognosis 70

Alive 5 years after operation:
Good prognosis..... 52 (63.4 per cent)
Poor or guarded prognosis ..... 21 (30.0 per cent)

To September, 1922—Total..... 85
Good prognosis..... 47
Poor or guarded prognosis 38

Alive 10 years after operation:
Good prognosis..... 22 (46.8 per cent)
Poor or guarded prognosis ..... 7 (18.4 per cent)

The results of treatment by complete and supravaginal hysterectomy and radium are shown in the following tables:

Complete hysterectomy:

Total to September, 1929..... 111
Operative deaths..... 9 (8.1 per cent)
Alive 3 years after operation... 71 (63.9 per cent)

Total to September, 1927..... 92
Operative deaths..... 7 (7.6 per cent)
Alive 5 years after operation... 52 (56.5 per cent)

Total to September, 1922..... 51
Operative deaths..... 5 (9.8 per cent)
Alive 10 years after operation 20 (39.2 per cent)

The nine operative deaths were attributed to the following causes: shock—3; pulmonary embolus—3; peritonitis—2; bronchopneumonia—1.

Supravaginal hysterectomy:

Total to September, 1929..... 47
Alive 3 years after operation... 26 (55.3 per cent)

Total to September, 1927..... 39
Alive 5 years after operation... 21 (53.8 per cent)

Total to September, 1922..... 24
Alive 10 years after operation 9 (37.5 per cent)

There were no operative deaths in this group.

Radium:

Total to September, 1929..... 23
Alive 3 years after operation... 7 (30.4 per cent)

Total to September, 1927..... 21
Alive 5 years after operation... 4 (19 per cent)

Total to September, 1922..... 10
Alive 10 years after operation..... None

Twenty-eight patients having complete or supravaginal hysterectomy received either pre- or postoperative treatment with radium. More often radium was used after operation, especially in those cases with a poor or guarded prognosis. Although it was effective in causing the disappearance of vaginal metastases, it could not be shown to affect the results. In the eleven cases which had received radium within six weeks before operation, the outcome suggested that the radium had been of value.

In a few cases of the group treated by complete hysterectomy, obesity, adhesions or the large size and fixedness of the uterus made it necessary to perform a supravaginal hysterectomy first and then to remove the cervix. Supravaginal hysterectomy was performed in a number of instances because the disease was apparently very early. More often it was necessitated because of technical difficulty (obesity, adhesions, immobility of the uterus) or because it seemed wise to subject the patient to as lit-

tle operative strain as possible (old age, diabetes, high blood pressure).

Radium alone was used either because operation was definitely contraindicated on account of senility or circulatory disease or because of excessive obesity with its greatly increased operative risk. It was also employed as a local palliative measure in some cases with hopelessly advanced cancer.

Discussion. Many of the younger patients with cancer of the endometrium had never been pregnant and had had an abnormally early menopause. The patients who had had children, on the other hand, in general had had their menopause between the ages of 48 and 56 and were much older when cancer developed. These factors and the finding of marked genital (including endometrial) atrophy, including obstructive conditions (intact hymen, vaginal atresia, cervical polyp, cervical stenosis), in the majority of cases indicate that this type of cancer is founded on a background of lack of function and poor uterine drainage.

In 1930 a patient well past the menopause was admitted because of staining. Careful examination and biopsies failed to reveal the source of trouble. A complete hysterectomy was performed and adenocarcinoma found with adenomyoma in the uterine wall, the endometrium being entirely atrophied. This immediately brought to mind another patient, included in the above series, who entered the hospital in October, 1926, because of bleeding after the menopause. Examination and biopsies were negative. In May and October of 1927 biopsies were again negative. Hysterectomy was not performed until November, 1928, by which time the uterus was practically replaced by adenocarcinoma. It would seem that this patient when first seen must have had cancer in an area of adenomyoma.

In the above series cancer was found sufficiently often in cases complaining of abnormal flowing at the time of the menopause to make it worthwhile emphasizing again that abnormal bleeding at this period is not by any means usually a functional upset and that every patient with this complaint deserves an examination under an anesthetic with biopsies of the cervix and endometrium.

Summary. Without discarding operative deaths or untraceable cases or making allowances for those that died of causes other than adenocarcinoma of the endometrium or of another type of cancer, the gross results of treatment in a series of 181 cases to September, 1929, are as follows: alive 3 years after operation—57.4 per cent; alive 5 years after operation—51.3 per cent; alive 10 years after operation—34.1 per cent. If only cases with a good prognosis at the time of operation are considered, the percentages become respectively 66.9, 63.4 and 46.8. (In compiling these last figures operative deaths, untraceable cases, and those dying of other causes were included.)

The operative mortality following complete hysterectomy ranged from 7.6 to 9.8 per cent. There were no operative deaths after supravaginal hysterectomy.

As would be expected, the results following complete hysterectomy were better than those following supravaginal hysterectomy, despite the operative mortality in the former. Although radium was not used extensively in this series, there was some evidence that it may be valuable as a preoperative measure.

It is interesting that there were no recurrences after the ten-year interval.

SUMMARY OF MORTALITY FROM AUTOMOBILE ACCIDENTS

Number of Deaths, April 19, 1931, to April 15, 1933, from Automobile Accidents

The Bureau of the Census announces that during the four weeks ending April 15, 1933, 86 large cities in the United States reported 513 deaths from automobile accidents. This number (513) compares with 560 deaths during the four weeks ending April 16, 1932. Most of these deaths were the result of accidents which occurred within the corporate limits of the city, although some accidents occurred outside of the city limits.

For comparison, the number of deaths due to automobile accidents within city limits is desirable. Such figures are available for the four-week period ending April 15, 1933, and for the corresponding four-week period of 1932 for all of the 86 cities, the four-week figure in 1933 being 421, as contrasted with 461 for the corresponding four weeks in 1932.

Considering by four-week periods since January, 1929, total deaths from automobile accidents, whether within the city limits or outside, the lowest total (482) appears for the four-week period ending February 23, 1929, and the highest (876) for the four-week period ending January 23, 1932.

For the 52 week periods ending April 15, 1933 and April 16, 1932, the totals for all the cities were respectively, 7,786 and 9,010, which indicate a recent rate of 21.3 per 100,000 population as against an earlier rate of 25.0 or a decrease of fifteen per cent in the rate during the year.

Eleven cities reported no deaths from automobile accidents for the last four weeks, while three cities reported no deaths from automobile accidents for the corresponding period of 1932.

For the last four-week period reports as to whether deaths occurred from automobile accidents within city limits or outside were received from all of the 86 cities reporting. In these cities in this four-week period, the total number of deaths from automobile accidents was 513 but only 421 of these were due to accidents within city limits.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19201

PAIN IN THE BACK AND LEGS

MEDICAL, SURGICAL, ORTHOPEDIC AND NEUROLOGICAL DEPARTMENTS

First admission. A New Englander fifty-one years old, a doorman, entered for the first time eleven years before his final admission complaining of vague right sided abdominal pain. Physical examination showed a large liver and free abdominal fluid. The urine showed sugar on three occasions. The blood sugar was 0.15 per cent. An abdominal tap yielded no fluid.

History of interval. He was followed in the Outpatient Department for a short while. Considerable sugar was found in the urine. Then he stopped reporting and did not reappear until two years later, when he came complaining of painful feet. No mention was made of glycosuria.

Second and third admissions. Six years and again seven years after his first entry he was treated in the wards for burns from Alpine lamp treatments for arthritis. Glycosuria was found both times.

History of interval. He did not report to the Outpatient clinic for his glycosuria, but was seen a number of times for blisters of his feet. Four years after his third entry one of these blisters was found to be infected. After two weeks of unsuccessful treatment he was referred to the wards.

There he gave a history of polydipsia and polyuria for many years. For five years his left great toe had felt numb. For a year he had had many blisters on his feet, usually requiring six to seven weeks to heal. For some months he had had cramps in his legs and pains in the sole of his foot. For nine weeks a blister on his left great toe had grown steadily worse. Two days before entry a large blister appeared after an application of heat to the foot. A urine test in the Outpatient Department showed sugar.

Fourth admission, four years after his third discharge.

Physical examination showed an obese man. The heart was normal except for distant sounds. The lungs were negative. There was tenderness in the right upper quadrant. The liver edge descended 2 to 3 centimeters on inspiration. There was no evidence of ascites. There was a deep punched-out ulcer of the left great toe and a large blister on the surface of the left foot. The left pupil was slightly larger than the right. Both were a little irregular. The reactions were normal. The ankle jerks were not obtained.

The urine showed a slight trace of albumin and a trace of sugar. The renal function was 20 per cent in the first fifteen minutes, 20 per cent in the second fifteen minutes. There was no fixation of gravity. Blood examination showed 6,900,000 red cells, hemoglobin 100 per cent, 8,300 white cells, 56 per cent polynuclears.

Under treatment the glycosuria was controlled and the ulcers healed well. He was discharged in nineteen days.

History of interval. At the time of discharge and at intervals for the next four months he had some very sharp shooting pain in the hip. Three months after discharge he began to have very sharp shooting pain in the lumbosacral and sacro-iliac regions, at times going up to the level of the angles of the scapulae. During the next month this pain was so severe that he was unable to sleep well. It was less on standing or walking and was aggravated by lying down. X-ray examination in the Outpatient Department showed hypertrophic arthritis of the lumbar vertebrae and sacro-iliac and hip joints. He thought Alpine lamp treatment lessened the back pain and entirely relieved the pain in the hip. Under treatment he had been sugar free. He had lost 22 pounds in seven months.

Fifth admission, four months after his fourth discharge.

On examination the heart sounds were of poor quality. Examination of the lungs was negative. His posture was extremely poor. The urine showed no sugar, specific gravity 1.012, a very faint trace of albumin at three tests. The blood sugar was 98.

With hot packs and exercises the back pain was much relieved. The patient was discharged in four days.

History of interval. For the following month the pain in the back and down the legs was much better. Then the patient slipped and twisted his back and following this had pain along the right costal margin and in the right upper quadrant radiating to the back near the tip of the right scapula, worse on lying down and persisting for the next month. He was following treatment for glycosuria but taking an almost normal diet. He urinated six times by day and six at night. He was gaining weight.

Sixth admission, two months after his fifth discharge.

Physical examination showed no important changes. There was questionable enlargement of the heart and moderate sclerosis of the artery walls. There was moderate tenderness on the right side of the abdomen from the nipple line to the midline and from the level of the fifth rib to 2 fingerbreadths below the costal margin.

The blood and urine were negative. Two Graham tests were negative. A lumbar puncture in the 4-5 lumbar interspace gave 15 cubic centimeters of clear colorless fluid, initial pressure 155, 28 red blood cells, some crenated, 4 lymphocytes, alcohol and ammonium sulphate positive, total protein 108, colloidal gold 0001123331, Wassermann negative. X-ray showed considerable reaction around the posterior ligaments at the lumbosacral joint. On reviewing the previous films it was seen that there was a destructive process involving the bodies of the second, third and fourth dorsal vertebrae with narrowing of the interspaces.

At the end of two weeks the patient was discharged, to return for further study.

History of interval. For two days after leaving the hospital the patient felt quite well. Then while motoring he had a recurrence of constant steady sharp pain starting in the epigastrium and extending to the right side. He also had at intervals sharp pain between his shoulder blades with a feeling of pins and needles in the same region. On the day before his seventh entry he had pain in the right side over a rib that was sore to touch and gave a feeling of pins and needles when he pressed it. There was numbness in the region of the abdominal pain.

Seventh admission, nine days after his sixth discharge. On examination he was rather poorly nourished. The right knee jerk was greater than the left. The left ankle jerk was absent, the right normal. No abnormalities of the heart or lungs were noted. Otherwise the examination was practically unchanged.

The urine showed a slight trace of sugar at five of seven tests. The blood sugar was 132. One observer noted that the urine had not been followed over a period of time while the patient was on an ordinary diet and that no glucose tolerance test had been done. A sugar tolerance test showed, fasting, 95 milligrams per 100 cubic centimeters, at one-half hour 125 milligrams, at one and one-half hours 202, at three hours 250.

All studies, x-ray, neurological and other, failed to show any cause for the pain except hypertrophic arthritis of the lower dorsal spine. Dr. James C. White advised against alcohol injection. At the end of three weeks the patient was discharged, to return for further study.

History of interval. The patient reported that three days before his seventh discharge he contracted a cough which persisted through the interval of seven days. The epigastric pain was worse on coughing. There was a little greenish sputum. He had taken only liquids for the past month because heavy foods aggravated the pain.

Eighth admission, one week after his seventh discharge.

On physical examination he was emaciated. He gave evidence of pain on slight motion of the body. The middorsal to the upper lumbar region of the spine was painful on any motion. He was unable to lie flat on his back. There was an indefinite area of tenderness on pressure over the lower dorsal and upper lumbar spine. The lungs were clear in front. The left base below the angle of the scapula showed increased breath sounds and bronchovesicular breathing. The right base showed diminished breath sounds with possibly increased spoken voice and dry râles. The right diaphragm moved less than the left. There was no tenderness. The abdomen was tender throughout, more so in the right upper quadrant.

Neurological examination. A somewhat uncoöperative and neurotic man with no disorientation or loss of memory. The cranial nerves were all normal. There was a questionable indefinite band 2 or 3 fingerbreadths wide of partial hyperesthesia and paresthesia passing around the right chest from the eighth and ninth dorsal spinous processes to just beyond the xyphoid. The motor nerves were rather weak but of equal strength on both sides. The knee jerks were present on reënforcement. The ankle jerks were very weak.

The urine showed a very slight trace of sugar on two examinations. The blood sugar was 192 to 108 milligrams. The white blood cell count was 11,750.

The temperature was 97.4° to 100.2°, the pulse 80 to 112, the respiration 18 to 28.

X-ray examination showed no change in the vertebrae and pelvis from the last examination. There had been a marked change in the chest. The heart and mediastinal contents were displaced towards the right. There was mottled dullness in the region of the right lower lobe which partially obscured the outline of the diaphragm. The right side of the diaphragm was elevated and limited in excursion. The remainder of the lung fields was clear. Two weeks later a further remarkable change was noted in the right side of the chest. Fluid and air were now present in the pleural cavity, with collapse of the lower lobe. Films taken of the lower dorsal and upper lumbar vertebrae showed lipiodol distributed in three irregular lines on the right side of the eleventh and twelfth dorsal and third upper lumbar vertebrae. There was no definite evidence of active disease in the lower

dorsal vertebrae. "All the x-ray findings in the spine could be explained by arthritis."

Two days after admission novocain injection was done. After it the patient said that the pain was gone from his back but was present in the epigastrium, apparently as a new pain. The patient complained much of weakness, yet coughed rather violently.

On the sixth day after admission novocain injection of the ninth and eleventh right dorsal nerves was done. The patient had no reaction or pain after it. He developed a characteristic zone of anesthesia and a hot, dry right foot. Two weeks after entry all the skin anesthesia had disappeared, but the patient still had an effective sympathetic block, as temperature measurements showed the average temperature of the right toes to be 7 or 8 degrees higher than that of the left toes.

On the twentieth day a chest tap was done in the right ninth interspace 7.5 centimeters from the spinous processes. About 20 cubic centimeters of purulent odorless fluid with some air (possibly a leaky connection) was removed; specific gravity 1.012, cell count 29,000, 86 per cent polynuclears, 14 per cent lymphocytes, occasional red blood cells.

It was learned from the patient's wife that his personality had completely changed since his illness. A change was now noted since he entered the ward. He was apparently becoming confused at times. He continued to fail rapidly and became cyanotic. His cough grew feeble. On the twenty-third day he died.

Clinical Discussion

Dr. Richard C. Cabot: "An abdominal tap yielded no fluid." That is strange, because just above we have "free abdominal fluid." It is possible that they managed to reduce that, or that while there was some fluid when he came in there was none when they did the tap.

The first six lines suggest cirrhosis of the liver more than anything else. We should have the same ascites in cardiac or kidney disease; but the chances of their saying so little of passive congestion and edema elsewhere would be less in either of those diseases.

Of course in any case in which we suspect cirrhosis and find that there is sugar in the urine, we put the two together and wonder whether they do not belong together in hemochromatosis, the pigmented type of cirrhosis. We shall have to see about that later.

In the report of the second and third admissions nothing is said about the liver or ascites.

Four years later he was referred to the wards, presumably because of his diabetes, which made the blisters difficult to heal. We do not hear nearly so much nowadays, under the better treatment, of the cramps that diabetes used to show, and also other evidences of neuritis, such as sciatica.

Of course as you know, gangrene often comes on with blisters that do not easily heal. They are expecting any time now that this will turn into gangrene.

"The ankle jerks were not obtained," so he may have neuritis extending down as far as that.

At the time of the fourth admission there was only a trace of sugar.

Whether the augmented number of red cells is of any importance or not we cannot yet tell. If he was somewhat cyanosed, as he easily may have been, it would not amount to anything. Nothing has been said about enlargement of the spleen or about the color in his face, which often goes along with a polycythemia. Polycythemia is a rare disease. We do not make the diagnosis unless we are driven to it.

So far we have nothing except a diabetes. There is no important difference between a chronic glycosuria and a diabetes. If we think it is not going to last we call it a glycosuria; if we think it is going to last we call it diabetes. This certainly has lasted. So he has a diabetes, and when that was not treated apparently he had these intractable ulcers due undoubtedly to arteriosclerosis in the leg arteries, with malnutrition aggravated by his glycosuria. Yet this diabetes has always been very slight, which is striking in view of its apparently being connected with arteriosclerosis in his feet.

As you know, what is still called "hypertrophic arthritis" is the normal senile process which begins when we are twenty-one and continues during life. You probably know the work done in the City Hospital in the Thorndike Memorial. They have looked into the knee joints of people dying from various causes and have found that this condition starts at twenty-one and gets worse at each decade. We call it hypertrophic arthritis when it produces symptoms. But most well people have it, though very few have symptoms from it. It is about as important as gray hair. As we see it in the vertebrae and other places it is a sign of age but nothing more. We have called it by various names and given it various etiologies, but the real etiology is age.

What the cause of his back pain was I do not know at all. I do not think there is any certain evidence that it was due to the so-called hypertrophic arthritis. There are many other structures in the back capable of giving such pain.

This was in the period when they were trying to find causes for so-called hypertrophic arthritis and looking at the gall bladder, as I think it is still fashionable to do.

Why they did a lumbar puncture I do not know. The report of it does not to me mean anything pathologic. We wonder why the protein is so high, but with all the negative tests here I do not see how we can call it anything. I do not know just what they mean by "reaction" in the report of the x-ray examination

THE MERIDEN HOSPITAL
1055
MERIDEN, CONN.

about the lumbosacral joint. I suppose they mean beginning calcification or ossification.

At the time of the seventh admission the blood sugar was 132. It has never been much higher. The sugar tolerance test shows that he has a lowered tolerance to carbohydrates.

"Hypertrophic arthritis of the lower dorsal spine" I do not understand, because the x-ray above said there was a destructive process, which of course is not hypertrophic. Such a process has been noticed a few times in diabetes, due to imperfect calcium balance.

The alcohol injections are to relieve pain.

Examination at his eighth admission showed him to be emaciated. That is quite different from anything we have heard before.

DR. AUBREY O. HAMPTON: He had numerous x-rays, not all of which are here, but as the report stated, practically the entire examination was negative except for hypertrophic arthritis, arteriosclerosis, and pathology in his chest. We have films of his gall bladder, his gastro-intestinal tract, his various joints, and I think he had an intravenous pyelogram. He had almost every examination we were capable of doing and they were all negative except for these findings. The calcification in his blood vessels is rather extensive, as we should expect in an elderly man with diabetes, perhaps not so extensive as usual.

His chest was normal at the examination during the seventh admission. Two months and about ten days later he had a very remarkable change in his chest. The right lower lobe had reduced in size, the diaphragm was elevated, and he had what we thought was evidence of partial collapse of the lower lobe. Eleven days later he had air and fluid in the right pleural space with collapse of the lower lobe and part of the middle and upper lobes. This air and fluid were quite definite and quite typical of hydropneumothorax or pyopneumothorax. We checked the dorsal vertebrae again, because at one examination we did think there was a destructive process here in the fourth and fifth. I think. After further study and more careful interpretation we decided that the appearance of destruction was produced by the absence of these huge spurs on one side.

With this story and the findings in the chest we tried to make a diagnosis. I happen to know that it was entirely wrong. We thought he had obstruction in the right main bronchus which produced suppuration in his lung, and the suppuration ruptured into his pleural cavity and produced the pyopneumothorax. The pain could have been due to metastases in the bone which we could not find because they had not been there long enough.

DR. CABOT: As I get it, then, our x-ray examination was negative. It did not show anything of importance. That destructive spinal process that interested me so much was not there at all. I heard Dr. Joslin talk about just such a thing last week, so I was interested to read about it here. But the last x-ray does not show it.

DR. HAMPTON: We were fairly positive about the chest findings.

DR. CABOT: So the one definite thing we have so far is water and air in the right side of the chest,—quite an unexpected event. In the chest tap they thought the air might have been due to a leaky connection. We shall come back to it after we have read the rest.

Here is a very long record with nothing in it that could suggest why he died until towards the end we get to this slight pyopneumothorax, of which I have no idea of the cause. He has had some pains, the origin of which I do not know. They may have had some connection with his spinal changes or may not.

DR. TRACY B. MALLORY: Here is his chart at the time he is supposed to have had the pyopneumothorax.

DR. CABOT: It is not much of a fever, is it? Yet it is rather hard, when people tell you they have taken pus out of a chest and an x-ray shows air, to deny pyopneumothorax.

DR. JAMES C. WHITE: Isn't that a pretty low specific gravity for an exudate? Perhaps some one had done the cell count wrong.

DR. CABOT: Yes, I think that point of Dr. White's is a good one. We should expect a higher gravity than this, certainly.

I do not make much of this case. I do not know what he died of. He starts in with a mild diabetes which looks as if it is going to be serious to his feet, but later his feet seem to get all right. Then he has these pains from which Dr. White delivered him, but I cannot imagine that these have any direct or important relation to his death. He has these bony changes which you heard described. He has had something in his chest. Aside from the x-ray there is no evidence of the physical signs which correspond with either air or fluid so far as I see. Haven't we any physical examination of his chest towards the end? It seems as though they ought to have committed themselves.

DR. MALLORY: Here is a chest diagram without a mark of any abnormality on it.

DR. WHITE: As I remember it I examined him at that stage. He had been raising a good deal of thick purulent sputum. There was definite but slight dullness to percussion in his right base, and there were some coarse râles, but no very definite change in voice or in breath sounds.

DR. CABOT: Ordinarily we do not pay much attention to the right base in a man with a big liver. We say his liver is sticking up there behind. So if we had to go on these physical signs alone we have no good evidence of disease above the diaphragm.

DR. WHITE: I do not think there is much doubt about there being fluid, because two or three of the men saw it.

DR. CABOT: They may have got the needle into a dilated bronchus, of course. They got only twenty cubic centimeters. That is not much to get out of anything else than a bronchus. In such a case one always thinks of pulmonary malignant disease. But we certainly have no physical signs of it or of any metastases anywhere else. I do not know whether he had a pyopneumothorax or not. On the evidence before this body it seems to me we cannot say whether he did or he did not, but in case he did it was insignificant. They may have got into a bronchus and taken some pus out when they tapped. He may have had a bronchiectasis, but that would not have killed him.

DR. WHITE: I was asked by Dr. Hodgson and Dr. Mixter to see this patient in connection with his pain. He had pain over the dermatomes where the arthritis was most marked. On the other hand, you see dozens of people with as much arthritis as he had without any pain at all. The thing we were considering, with his loss of weight, anemia, and general appearance, was malignant disease, either primary or metastatic in the spine or spinal cord. That is why the lumbar puncture was done. It showed a high protein and nothing else. When no diagnosis could be made, they asked me to try to inject alcohol into those middorsal sympathetic nerves to see if I could relieve the pain. We have demonstrated pretty well that with novocain and then with alcohol we can effectively block sympathetic ganglia and sympathetic rami. We have had one case of angina pectoris where the freedom from pain has lasted five years. On the other hand, you cannot block cerebrospinal nerves for any length of time. After you insert your needle to the lower border of a rib you push it several centimeters further, so that its tip lies in close contact with the paravertebral sympathetic trunk and its rami communicating with the intercostal nerves. The needle is then just below the intervertebral foramen and therefore novocain or alcohol will infiltrate the intercostal nerves and go down to the corresponding sympathetic ganglia. If you do that accurately you produce a zone of skin anesthesia corresponding to the distribution of the given intercostal nerves, and you also produce characteristic signs of sympathetic paralysis in the region. That is to say that at this level you would block the vasomotor fibres to the leg and produce some paralysis of the sweat glands as well. The sensation in the intercostal nerves would come back in about two weeks or sometimes sooner than that, but the sympathetic paralysis should be permanent. So if his pain were due to a focus within the meninges or in the intervertebral disks, which are innervated by the sympathetic, you ought to get permanent relief. If, on the other hand, this pain were due to an arthritic spur impinging on an intercostal nerve, you would not get permanent relief. I therefore tried a novocain injection first to see if the pain could be relieved, and it was, for two hours. A few days later I repeated the novocain injection and then injected 95 per cent alcohol. Following that he never had any more back pain. He still had some epigastric pain, but this was less troublesome. The alcohol injection produced no neuritis and he remained a good deal more comfortable. I know the alcohol was in the right place because I injected a few drops of lipiodol with it, and x-ray plates showed it to be just where it ought to be. He also had a warm foot which remained eight or nine degrees warmer the rest of his life. I do not believe the injection had anything to do with the condition in his lung.

DR. HAMPTON: The changes in his lung were noticeable before you did the injection.

DR. WHITE: He was raising purulent sputum before the injection.

DR. CABOT: I want to ask some conundrums of Dr. White. In old days, when Dr. Osler's textbook was the only textbook there was (as I think it ought to be still), when it came to the section on lumbago, which as you know is an unfashionable diagnosis nowadays, Osler had an extraordinary treatment which he was most enthusiastic about and which he could generally not get anybody else to try. It was to take hatpins, such as ladies used to wear, and stick them deep into the lumbar muscles. He said it gave extraordinary relief. I tried it on some men in the Outpatient Department here, but I could not tell whether it did good, because they never came back. As Dr. White was talking I wondered whether if he put his needles in and then took them out without injecting anything, it would relieve the pain.

DR. WHITE: If you put in novocain alone it gives relief for only a few hours, because as soon as the novocain is absorbed the pain usually returns. I can imagine putting a needle in and producing a hematoma which would act as a foreign protein, produce fever, and thereby affect the pain. On this or on a psychogenic basis inserting needles alone might be of benefit. It is also true that occasionally novocain alone will give long-lasting relief.

DR. CABOT: This was one of the jokes about Dr. Osler. He had very little therapeutics in his book. He said, "Let nature do the work." But I have always wondered whether acupuncture, which used to be very popular in the years prior to Osler, had not more in it than we realized.

A lame and impotent conclusion is all I can add. I do not know what this man died of. I am pretty sure he did not die of any of the things we have been considering, but I do not know where to look for any forgotten latent process. If he had malignant disease I cannot

say where. At one time it looked as if he had it in those second to fourth dorsal vertebrae, but later x-rays did not show it there or anywhere else. I think he may have had bronchiectasis, but I do not think he died of it. There is no evidence that he had a bad heart. So it is with his kidneys. We have not much evidence against them. So if we do our duty here I should say: on the evidence of the facts presented to us, no diagnosis, no cause of death is justifiable.

Having committed myself I should like to hear from anybody else.

A STUDENT: Doesn't tuberculosis in diabetics make itself known?

DR. CABOT: I have seen tuberculosis in the childhood form of diabetes. The trouble is we have no diabetes to speak of. I have never seen fatal tuberculosis complicating diabetes of so mild a type. This man is emaciated. Can he have tuberculosis anywhere else? I cannot make the diagnosis of tuberculosis because I cannot say where it is.

A STUDENT: How do you account for the red blood cells in the spinal fluid, some of them crenated?

DR. CABOT: I think they were due to trauma in doing the puncture.

A STUDENT: Do not the change in personality and the high protein in the spinal fluid have to be explained by some pathology? That sounds like frontal lobe pathology.

DR. CABOT: No, I do not think that such a change is nearly enough evidence for a diagnosis of brain tumor or brain hemorrhage or any other change that I can name. I do not think we have nearly enough evidence to say that he had cerebral arteriosclerosis causing important or fatal symptoms. Such a personality change comes on in many elderly people; it seems to me that we do not need any other explanation than his age.

A STUDENT: Would you consider a subdiaphragmatic abscess perforating the pleural cavity?

DR. CABOT: No; I should say that the x-ray men would have shown us that. The diaphragm would have been fixed, and it was not.

A STUDENT: Wouldn't central nervous system neoplasm explain the pain?

DR. CABOT: I should say no. We will ask Dr. White about it.

DR. WHITE: A spinal cord tumor might, but a brain tumor would not account for anything except his headache.

DR. HAMPTON: A metastatic brain tumor would explain it.

DR. WHITE: Of course abscess of the lung is very likely to have metastasis in the brain. We could put the two together in that way.

A STUDENT: Would the diabetes be adequate excuse for a large liver?

DR. CABOT: I do not think it is fair to say he has diabetes. Bronzed diabetes is always in one's mind when we have diabetes and a large liver. But I do not think we have any adequate evidence of bronzed diabetes or of any other kind of diabetes as the cause of this man's death.

A STUDENT: Then you have no explanation of the big liver.

DR. CABOT: No, and I do not think there is much evidence that it was big, except in the beginning.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Possible carcinoma of the lungs with possible metastases to the brain and spinal cord.
Possible secondary lung abscess.
Diabetes.

DR. RICHARD C. CABOT'S DIAGNOSES

No evidence of a cause for death.
Probably some bronchiectasis, but nothing of importance.

ANATOMIC DIAGNOSES

Bronchiectasis, moderate, right middle and lower lobes.
Acute purulent tracheitis, bronchitis and bronchiolitis.
Chronic endocarditis, mitral, possibly rheumatic.
Arteriosclerosis, moderate aortic, marked renal.

PATHOLOGIC DISCUSSION

DR. MALLORY: I have very little more to add, for we found only one thing in this man. He had a rather definite diffuse bronchiectasis of the smaller bronchi in the right lower and middle lobes. The largest cavity that we found at the time of autopsy was not much over one centimeter in diameter. On the other hand it is quite possible that during life they might have been considerably bigger than that, and I think that the twenty cubic centimeters of pus which was drawn from the chest probably did come from one of the dilated bronchi. It is perhaps a little surprising that they were able to put a needle through a normal pleural cavity into the bronchus and bring it out again and not infect the pleura, but it did not show any lesion.

All his other organs were essentially negative. The heart and kidneys were of normal size. The liver seemed a little large, 1900 grams, but showed nothing abnormal. The brain and cord were normal. There was a very marked hypertrophic arthritis.

DR. CABOT: You do not think that is cause enough for death, do you?

DR. MALLORY: I have seen people die before in whom I could find nothing but bronchiectasis. I think they have usually been more obviously sick from it than this, have had a clinical condition that came close to pneumonia. Only at autopsy are we able to say that the in-

fection is still in the bronchioles and has not spread into the surrounding tissue.

DR. HAMPTON: How long do you think he had had bronchiectasis?

DR. MALLORY: It does not seem as though he had had it very long,—a year at most.

DR. HAMPTON: We have a film here taken three months before death.

A STUDENT: What caused the personality changes?

DR. MALLORY: I do not think one ever needs to be surprised about that in people over the age of fifty, particularly when they have obvious arteriosclerosis.

A STUDENT: Do you think he had diabetes?

DR. MALLORY: It might I suppose be called diabetes, the very mild diabetes that rather old people have, that goes with moderate arteriosclerosis, that is not progressive.

A STUDENT: The result of arteriosclerosis?

DR. MALLORY: I do not know whether or not it is a result. They are very often concomitant. A large percentage of people over sixty may have a slightly elevated blood sugar without ever developing real progressive diabetes.

DR. CABOT: And they get along perfectly well.

A STUDENT: Do you think the ulcers on the feet were due to something more than the diabetes? Were they due to a combination of mild diabetes and arteriosclerosis?

DR. WHITE: Of course he did not show any particular trophic changes in the feet when he was in the last time. If he had severe arteriosclerosis of his peripheral vessels he should have had congestion of his toes when he was standing. Anyone who has arteriosclerosis severe enough to produce actual gangrenous changes in the toes shows marked cyanosis in the feet when they are dependent, and that was not true of him. We do see people with diabetes who get into trouble with superficial skin infection, but they do not show the characteristic changes of arteriosclerosis. They have a perfectly good peripheral circulation, but a diminished resistance of the skin to infection. Very often they have an anesthesia of the toes, so that they develop trophic changes. When I saw him he had no blisters at all and he had no anesthesia of the feet. All we have to go on is the history of these ulcers, which I am at a loss to explain as I never had a chance to see them.

DR. CABOT: A former pathologist of this hospital used to write down about once or twice a year, "*Causa mortis ignota.*" I thought it was a good custom.

DR. MALLORY: I fear the trouble is that if we were candid about it we should write it down in about fifty per cent of the cases.

DR. CABOT: I think science would advance faster if we did. To show the holes in our knowledge of which we are aware and to show them clearly—I think we are not so eager about that as we should be. Bronchiectasis is all that we found in this man, and we all know that people can live for years with bronchiectasis. Three months before his death we had an x-ray which showed nothing in his lung. That is not conclusive, of course, but it is conclusive for an extensive process. I think this case should be signalized, because it is a more striking example of the limitations of present medical knowledge than any we have had for a long time. I should be glad if you would argue on the other side, Dr. Mallory.

DR. MALLORY: I think the only argument there is whether or not it is possible for a man to die because he does not want to live. I think we very often see something very closely resembling that. We have nothing definitely bearing upon the point in this case, although the man's history suggests it. In surgery we see such cases often. Some patients will pull through the most terrific complications, having an intense desire to live, while the next person, in whom we cannot find anything definite, will slowly go down hill and die.

DR. CABOT: I am a strong believer in what Dr. Mallory has said.

CASE 19202

THORACIC SINUS OF THREE YEARS' DURATION

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. CHAMP LYONS*: This is the case of a forty-two year old Scotch express agent. We have various stories of hospitalization. It is well to go back to the beginning.

Six years before entry he was in another hospital complaining of epigastric pain of five years' duration. A partial gastrectomy with local excision of a duodenal ulcer was done. He was symptom-free after the operation.

Four years later he reported at the same hospital with a lump in his right chest of eighteen months' duration dating from an injury. The lump was about the size of a pea when first noticed and gradually increased to about the size of a small lemon. His physician made an unsuccessful attempt at aspiration. Biopsy was attempted. The specimen was a gritty, gelatinous substance which was reported as a mixed tumor of the breast, non-malignant. He was discharged with a diagnosis of osteochondrosis of the sixth and seventh ribs. There was a persistent draining sinus for which ultraviolet treatment was instituted without benefit.

Eleven months later he reported at a second hospital with clubbed fingers, persistent nonproductive hacking cough and a draining sinus. His sputum was negative for tuberculosis. X-ray revealed obliteration of the right diaphragm and

*Junior interne on the West Surgical service.

the costophrenic angle. This was interpreted as encapsulated empyema. He was advised to enter the hospital, but refused.

There is no further history until a year and a half later, when he entered the Emergency Ward of the Massachusetts General Hospital complaining of fatigue, persistent cough at times productive of foul yellowish sputum, and a persistent draining sinus. We felt he had an encapsulated empyema. This was confirmed by x-ray and the Thoracic Clinic.

On examination the right pupil was smaller than the left. The right chest did not move. The right base presented signs of fluid. Several tappings of the chest were unsuccessful. The last one yielded 10 cubic centimeters of blood, we thought probably attributable to our having entered a vein. A biopsy of the persistent sinus was undertaken by Dr. Cope.

We presented the situation to the patient's family and they accepted it. Dr. Churchill agreed to operate.

Dr. George W. Holmes: There is dullness at the right base without much displacement of the heart. The upper border of the dull area is more or less horizontal, and might correspond to the edge of the lobe or to a fluid level. In the lateral view it is roughly triangular in shape. The shadow of the diaphragm on the opposite side is visible above it. One would not expect to find any large amount of fluid. If it was fluid it would have to be encapsulated. I do not think there is anything very characteristic of malignant tumor in the x-ray picture.

The film of the ribs is not here. We ought to have it. The report is: "There is considerable irregularity and fuzziness in the outline of the 6th, 7th, 8th, 9th, and 10th ribs. The appearance suggests a periosteal reaction."

Clinical Discussion

Dr. Lyons: At biopsy, after removing from the sinus some fluid which showed non-hemolytic streptococcus Dr. Cope broke into a mass of gritty material which was later reported as an osteogenic sarcoma of the chondrosarcoma type.

Dr. Edward D. Churchill: This was a border-line surgical problem, because the tumor had already been opened a year previously and the patient had a secondarily infected draining sinus. The diagnosis of malignant chondrosarcoma was definitely confirmed, so an attempt at operative removal seemed justifiable.

Intratracheal ether anesthesia was employed. It was very difficult to determine whether the tumor could be removed or not. Very little hemorrhage was encountered. During the operation the patient suddenly became cyanotic and shortly thereafter died due to the production of a bilateral pneumothorax. The pneumothorax on the right side was formed during an attempt to separate the tumor from the anterior mediastinum. Even with positive pressure ventilation a small tear into the opposite pleural cavity may be fatal if a valve-like opening allows air to enter but prevents its escape. No matter how much positive pressure is maintained within the trachea it will not expand a lung within a closed pneumothorax or one with a valve-like opening.

The proper treatment in such an emergency is either to open the other pleural cavity wide and allow the air to escape as the lung is expanded by the positive intratracheal pressure or to put a needle into the pleural cavity and withdraw the air with suction.

The tumor itself was inoperable and much more extensive than the x-ray picture had led us to believe. To have removed it would have meant excision of practically the entire left leaf of the diaphragm.

Clinical Diagnoses (from Hospital Record)

Chondrosarcoma, chest wall, right.
Circulatory failure?

Anatomic Diagnoses

Osteogenic sarcoma (chondrosarcoma) of the ribs with invasion of the right lung and possible extension to the liver.
Operative wounds: resection for removal of rib tumor; old cholecystectomy.
Bilateral pneumothorax.
Mediastinal emphysema.
Secondary empyema.
Hypertrophic pulmonary osteoarthropathy.
Arteriosclerosis, slight, aortic.
Slight hypertrophy of the median lobe of the prostate.

Pathologic Discussion

Dr. Tracy B. Mallory: This is the second pleural cavity tumor we have had recently in which autopsy has shown that the tumor was two or three times more extensive than had been suspected on x-ray or other clinical grounds.

The specimen shows an enormous tumor arising from the ribs and firmly adherent to the lung on one border, to the diaphragm on the other. Removal would have required resection of three or four ribs and practically all the diaphragm, and it is doubtful if the tumor could have been freed from the lung even then. We were unable to dissect it free from the lung at the time of autopsy without actually tearing into the lung tissue.

Microscopic examination shows a very well differentiated cartilaginous and bony tumor.

The tumor would have to be classified as an osteogenic sarcoma, though it is obviously a very slowly growing one and most of the differentia-

tion is towards cartilage with only here and there a little bone formation. It is the type of tumor in which metastases would be expected to be very late, but almost inevitable eventually. The proportion of cured cases even in these relatively very well differentiated tumors is a very small one, not much over 15 per cent probably.

DR. CHURCHILL: Did that tumor go through the diaphragm and invade the liver at all?

DR. MALLORY: No, but it was so firmly adherent to the diaphragm that it could not have been freed.

DR. CHURCHILL: The long standing inflammation from the secondary infection probably caused the formation of the adhesions.

DR. MALLORY: I think that is true as far as the adhesions to the lung were concerned. Above the tumor we did find a little pocket of pus, evidently the remnant of a very old empyema. The tumor itself, although firmly adherent to the lung, did not seem to be actually invading it.

DR. HOLMES: Was there any invasion of either lobe of the lung?

DR. MALLORY: No.

DR. HOLMES: Was there extension along the interlobar fissure?

DR. MALLORY: No.

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SIMPLIFIED INFANT FEEDING

INFANT feeding, as the years have passed since first percentage mixtures put this art on a scientific basis, has swung far around the circle in the direction of increased simplicity. Roteh's methods, excellent as they were, though complicated, convinced many a medical student of two decades ago that pediatrics was not for him; the success of whole milk dilutions, their calories bolstered up by the addition of sugar in one form or another, came as a welcome relief to a perplexed multitude of embryonic physicians. Boiling assured bacteriological safety and increased the digestibility of these simple mixtures.

Further complications were introduced when hydrogen ion concentration was recognized, and for several years lactic acid was enthusiastically added, drop by drop; other acids were seized upon in the ardor for experimentation; hydrochloric, and even lemon juice. Commercial manufacturers, quick to follow every lead, have maintained a steady stream of prepared foods, liquid, concentrated and dried, and the modifying sugars are now legion,—and vitaminized.

It is interesting to note that unsweetened evaporated milk, long damned because of its familial association with sweetened condensed milk, the latter obviously unsuited, although once widely advertised for infant feeding, is coming into its own. Here we have, pari passu, about the simplest, frequently the most convenient, one of the cheapest and very nearly the safest of all foods. Simply add sugar and water (in the correct proportions) and serve!

AN AID TO PHYSICIANS

JUDGING by the numerous requests for information received by the *Journal* from time to time, the opinion that there is abundant need of a compilation of the community resources relating to medicine and allied interests in and about Boston is justified.

To meet this need a physician's handbook has been made in which are set forth in available form the legal responsibilities incident to medical practice and the diagnostic, therapeutic and social resources needed for the guidance of doctors in dealing with illness. This publication under the auspices of the Massachusetts General Hospital has been made possible by an anonymous donor of a fund. The work of collecting and arranging the facts has been done by a committee consisting of John P. Monks, M.D., Chairman, Josephine Barbour, George H. Bigelow, M.D., Ida M. Cannon and Dwight Siscoe, M.D.

There will, of necessity, be changes and additions in future editions, and communications which will aid the committee in the work should be sent to Miss Ida M. Cannon, Chief of Social Service, Massachusetts General Hospital.

This pamphlet should be in the library of every physician and social worker in Boston. It may well be the vade mecum of the young practitioner, for its perusal will inform him of his legal obligations.

Copies may be secured at the Massachusetts General Hospital.

Massachusetts Medical Society

THE COMING ANNUAL MEETING

THE meeting of the state medical society June 5, 6 and 7 promises to be of more than average interest to the general practitioner. Aside from the programs of the six Sections there are to be seventeen scientific exhibits as well as thirty-

three displays by commercial firms. The showing of the cranial nerves with motion pictures in colors by Dr. Truesdale of Fall River in the ball room on Monday morning, June 5 should attract a large audience. Dr. Truesdale has expended a large amount of time and money in the preparation of this instructive demonstration.

Put these dates in your engagement book so that there will be little possibility of interference with attendance at the exercises.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

KEEFER, CHESTER S. B.S., M.S., M.D. Johns Hopkins University 1922. Assistant Professor of Medicine, Harvard Medical School. Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital. His subject is "The Classification and Certain Pathological Aspects of Chronic Arthritis." Page 1027. Address: Thorndike Memorial Laboratory, Boston City Hospital, Boston.

DAWSON, M. H. M.D. McGill University Faculty of Medicine 1923. Assistant Attending Physician, Presbyterian Hospital, New York City. Assistant Professor of Medicine, College of Physicians and Surgeons, Columbia University. Address: 620 W. 168th Street, New York City. Associated with him is

BOOTS, R. H. M.D. University of Pittsburgh School of Medicine 1915. Assistant Attending Physician, Presbyterian Hospital, New York City. Associate in Medicine, College of Physicians and Surgeons, Columbia University. Address: 791 Park Avenue, New York City. Their subject is "Recent Studies in Rheumatoid (Chronic Infectious, Atrophic) Arthritis." Page 1030.

BAUER, WALTER. B.S., M.D. University of Michigan 1922. Associate Physician, Massachusetts General Hospital. Assistant Professor and Tutor in Medicine, Harvard Medical School. Address: Massachusetts General Hospital, Boston. Associated with him are

BENNETT, GRANVILLE A. B.S., M.D. University of Iowa 1925. Instructor in Pathology, Harvard Medical School. Address: Harvard Medical School, Boston. And

SHORT, CHARLES L. A.B., M.D. Harvard University Medical School 1928. Chief Resident, Massachusetts General Hospital. Address: Massachusetts General Hospital, Boston. Their subject is "Speculations on the Etiology of Rheumatoid Arthritis Based on Physiological Studies of Normal Joints." Page 1035.

KIEFER, EVERETT D. A.B., M.D. Harvard University Medical School 1926. Physician, Department of Gastro-Enterology, Lahey Clinic. His subject is "Tumors of the Small Intestine." Page 1042. Address: 605 Commonwealth Avenue, Boston.

SMITH, GEORGE V. A.B., M.D. Harvard University Medical School 1926. Assistant Visiting Surgeon, Free Hospital for Women, Brookline, Mass. His subject is "A Brief Summary of Results in the Treatment of Carcinoma of the Endometrium." Page 1049. Address: Free Hospital for Women, Brookline, Mass.

BOSTON MEDICAL LIBRARY

THE SWIMMER MANUSCRIPT, CHEROKEE SACRED FORMULAS AND MEDICINAL PRESCRIPTIONS BY JAMES MOONEY AND FRANZ M. OLBRECHTS

A RECENT acquisition of the library is the above entitled monograph issued from the Smithsonian Institute. It is sponsored by the Bureau of American Ethnology and should be of interest to students of American Medical History. As a result of much painstaking research, carried on in the field through interviews with the medicine men of the tribe, these formulas for the treatment of various diseases have been deciphered. They are of considerable importance and interest, not only to physicians but to all concerned in the study of the reactions of the primitive mind, for this is not merely a study of the therapeutics of the untutored savage but is quite as much an inquiry into the processes of deduction practised by untrained intellects. Mooney, who spent his life among these people, living with them so long that he gained their confidence and affection, died before he had completed his task. It was taken up by Olbrechts who discovered that the original Cherokee manuscript, which Mooney found, had been lost and he was obliged to ingratiate himself with these people before he could continue the study. Many of those who had been most helpful to his predecessor had in the meantime died. The difficulties of the language are great but, in particular, the interpretation of the ceremonial dialect in which the medical formulas were expressed was known to only a few and among them, even, there were many disagreements.

As a basis for an understanding of the methods of treatment and the raison d'être for their employment by the tribal medicine men, the research takes up the causes of disease as recognized by the Indian, for until this is appreciated, it is impossible to understand their processes of reasoning.

To the mind of the Indian medicine man no one ever becomes ill without cause and with but few exceptions the individual is blamable. He has transgressed some "taboo," has had a bad dream, has been the subject of the machinations

of certain "Little People," who have become angry with him because of slights, abuses, lack of respect or any other motive. Among these supernatural causes may be mentioned spirits, as for example, the sun, the moon, the river, fire, thunder and various "Little People," fairy-like in their description, who dwell in the rocks and forests and speak the Cherokee language. There are also animal spirits, prototypes of the common animals who are never seen or heard but are the avengers of the hunters who destroy those creatures of which they are the prototype. Ghosts, both human and animal, may be the cause.

Besides these supernatural causes, there are "disease causes" sent by fellow human beings, as for example, witches, "man killers" and women during their menstrual periods. In regard to contagious diseases the Indian has cultivated the idea that epidemic diseases are an infliction of the white people for purposes of revenge or personal gain and they have in the language a generic term for contagious disease which means "he (the 'disease causer') drives it (the disease) about". With such information as is supplied in detail in respect to these disease causes it becomes quite apparent that the mind of the medicine man, acting on the data obtained from a patient, who, himself, is often well versed in the tribal medical lore, is surprisingly logical in its reasoning. The Cherokee does not trouble himself much about prognosis but resorts to divination by "the beads" when he ventures upon a determination of the outcome of a particular illness.

The materia medica of the Cherokee is for the most part botanical though he has some faith in remedies of animal or vegetable origin. The basis upon which such remedies as they prescribe is founded is rarely a matter of experience but rather mythological. In collecting the herbs, roots and barks which they use, they are influenced by symbolism in the appearance of the tree or bush as likely to have a bearing upon the therapeutic purpose they have in view. The selection of lightning-struck wood, bark from the sunny side of a tree and the roots of an inverted raspberry branch, i.e., the roots from a "runner" of this shrub, indicate the influence of the occult in their therapeutic conceptions. The collection of the simples, their preparation and method of administration were all connected with more or less ceremonial.

After the collection of the simples used in the treatment of a given case, and except for those used at childbirth, they are never kept, but gathered fresh, they are crushed and a decoction or infusion made. The drugs are administered usually by the medicine man himself though at times he may delegate this office to a member of the family and the dosage is generally "as much as he can hold". As regards the animal sources of their remedies it appears that certain characteristics of the creature from which they are obtained influenced their selection, as for example, the oil obtained from eel skins is credited with power to cure rheumatism because the eel is a lithe and supple creature. Stones and flints are often boiled along with herbs because it is thought they will "cut the disease to pieces." Certain medicines are administered by blowing them over the patient with a hollow blowing tube and much importance is attached to the use of gourds, shells, beads and rattles as accessories to the treatments. Massage, inunction, accompanied by scarification, sweating in a "hot house", and vapor baths followed by a plunge in a stream of cold water are employed extensively. Emesis is induced in certain contagious diseases and the patient is made to vomit into the river in order that the disease may be carried away or transferred to others. Diet and many taboos figure prominently in many of the formulas directed to the cure of disease. With notable frequency the use of hot food and salt is taboo. Their surgery is even more crude than their medicine and considerably more restricted in its scope. Scarification, splinting, of a rough sort, in fractures, buzzard's down and spider's webs as stypties in hemorrhages and the "sucking horn," a kind of cupping glass, as a counter-irritant, various decoctions or infusions for boils, wounds, etc., constitute the bulk of their surgical practice. Prophylaxis, except in certain symbolical forms, is practically neglected, even the medicine dance which was formerly resorted to in warding off an epidemic is not often indulged in now. When a Cherokee recovers from a serious illness, it is probably to be ascribed more to his strong constitution and the healthful environment in which he lives, than to any specific therapy followed in the treatment of his disease. The medicine man among the Cherokee is the most dominant figure in tribal life, exercising an influence in many of its affairs outside the realm of medicine. There are three different orders among them, although in a few individual instances all three have been united in one. They may be classed as "priests", "magicians" and "divinators". Their knowledge of tribal lore and ceremonial usage is complete and their acquaintance with botany is often quite extensive. Some of the medicine men whom Mooney met were familiar with from 150-200 herbs and plants. The qualification for practice as a medicine man is acquired after much prolonged and trying study and testing. Though the office is not an hereditary one, still the taste for it seems to run in families for generations. Many interesting details of tribal views regarding pregnancy, contraception, abortion, parturition, the care and rearing of children, death and burial, the soul and after-life are related interestingly. Following this preliminary survey of the Cherokee's views of life and particularly his attitude toward disease, a considerable number of the

formulas employed by the medicine men in trying to serve their people are recounted. The formulas are either sung or recited by the medicine man and adhere pretty closely to a stilted form, beginning with (1) an exclamation of warning to attract the attention of the spirit addressed; (2) naming the spirit and giving his color or his place of abode; (3) a remark extolling the spirit; (4) statement of the cause of the disease; (5) an uncomplimentary reference to the "disease causer"; (6) a reason for the spirit to give his aid; (7) a statement, emphatically expressed, to the effect that relief has been accomplished and (8) finally, an exclamation, the same or similar to that with which the formula opened. A few of the titles of these formulas are selected at random; "This is to cure when they have them itching, a genital pruritus." "This is when they are sick with the yellow urinary tract disease." "This is when they have them drooping," an ocular affection in which the patient cannot bear the light. "This is to treat them with when he habitually breaks them," i.e., rheumatism. "This is to treat them when they have blisters," etc.

With each of these formulas, following the general plan of construction, the herbal remedy is given and directions for its preparation and administration are supplied together with the appropriate ritual.

MASSACHUSETTS LEGISLATIVE NOTES

BILLS WHICH HAVE NOT RECEIVED APPROVAL BY THE LEGISLATURE

S. 124—To secure payment of hospitals for services rendered in connection with motor vehicle accidents.

H. 519—Petition of Henry M. Landesman relative to liens of physicians, nurses and hospitals for services rendered in treatment of injuries for which patients receive compensation.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

The Prevention of Diabetes*

BY DWIGHT L. SISCOE, M.D.

Diabetes most frequently develops in the fourth and fifth decades of life and occurs as frequently in men as in women. It is a disease which results from failure of the pancreas to manufacture enough insulin to completely digest and utilize the food consumed. The exact method by which insulin is formed and exerts its influence is not yet known; therefore, no definite preventive measure, which strikes at the real root of the disease, can yet be formulated. We do know, however, several predisposing factors, the control of which will greatly reduce the incidence of diabetes. Chief among these factors are obesity, heredity and gall stones.

There is definite need, in the United States, for the prevention of diabetes, because, according to figures collected by insurance companies, there are over two million diabetics in the United States[1]. In the health surveys made in Framingham[2] and Winchester[3], Massachusetts, 1.7 per cent and 1.6 per cent, respectively, of the people were found to have diabetes. These figures, if applied to the entire United States, indicate the presence of about two million diabetics. In this country the number of cases trebled between 1880 and 1900 and doubled between 1900 and 1920[4]. The lengthened span of life increases the opportunity for the development of diabetes, because the disease most frequently develops in people about fifty years of age. Further indication for the need of prevention of diabetes lies in the fact that many people erroneously assume that insulin is a "*cure*," available to them if they develop the disease. Another pertinent fact is that the per capita consumption of food is greater in the United States than in any other country[5]. It is astonishing to note that the per capita consumption of sugar in the United States has steadily increased from 66.6 pounds in 1900 to 98.5 pounds in 1931[6]. The mortality rate from diabetes in New York City in 1932 was 29.3 per 100,000 persons, greater than in any other city in the world.

Diabetes is one of the great penalties of obesity. At least 90 per cent[3] of diabetic people have been overweight before the development of diabetes, and approximately 80 per cent of them have been at least 10 per cent above their normal average weight. Diabetes occurs seventy-nine times more frequently in people 21 per cent overweight than in people 21 per cent underweight[7]. In other words, if you weigh 180 pounds instead of your normal 150 pounds, your chance of developing diabetes is seventy-nine times greater than if you weigh only 120 pounds, 20 per cent under your normal weight. Diabetes does occur in people who are definitely below their normal weight, but this is comparatively rare and is usually associated with heredity. The Jewish race seems unusually susceptible to diabetes, possibly because they are especially prone to obesity; however, some other still unknown factor is probably present. It has been shown that one out of every twelve obese Gentiles develops diabetes, whereas one out of every eight obese Jews develops the disease[8]. We do not know the reason for this greater incidence among the Jews.

Wherever conjugal diabetes has been found, i.e., present in both husband and wife, each has been overweight and the cause has been aptly stated as "exposure to good food rather than to one another"[9].

In view of these facts, the first principle in the prevention of diabetes is the prevention of obesity,

*Station WBZ, March 21, 1933, 4:30 P.M.

and this means briefly less food and more exercise. An abstemious diet and adequate exercise may, therefore, prevent diabetes. Limitation of the total quantity of food eaten is more important than restriction of specific kinds of food. In their effort to lose weight, some people do not eat anything all day in order that they may have the pleasure of eating a huge meal in the evening; and this foolish custom may even help precipitate the onset of diabetes by putting too large a digestive load on the pancreas all at once. Diabetes results not only from eating too much sugar and sweet food, but from overeating *all* kinds of food. In the treatment of diabetes, we have found undernutrition of great value. Consider, therefore, how much more valuable it may be as a preventive measure.

Fat people who develop diabetes usually do so when they are about 50 years old. The development of this type of diabetes is slow and insidious and is characterized by the intermittent appearance of sugar in the urine. Hence, prevention and control are possible, provided the first appearance of sugar in the urine is heeded. Every person should have the urine tested at least once each year and after 40 years of age, especially if obesity or family tendency to diabetes is present, once every six months. If you would prevent diabetes in yourself, maintain your average normal weight and, after 35 years of age, be five or ten per cent below this weight, and in addition, have the urine tested once or twice each year.

The part played by heredity in the development of diabetes has occasioned much discussion in medical circles, but most authorities agree that there is a definite familial tendency to the disease. In about 26 per cent of all cases of diabetes, an hereditary or familial tendency has been demonstrated[10]. A silver lining to this cloud, however, is the fact that diabetes of hereditary origin is frequently very mild in type[11].

Those people who have an hereditary tendency to diabetes may counteract this tendency by avoiding obesity. Overindulgence in any sweet food may be the determining factor in such a person[12]. It is wise for him to avoid very sweet things, such as candy, maple sugar, syrups, rich preserves and very sweet pastries. He should make a special effort to keep himself in the best possible physical condition, should get plenty of outdoor exercise and adequate mental relaxation. He should never fail to have the urine tested twice each year and, above all things, should never allow himself to become fat.

Persons with disease of the gall bladder not infrequently develop diabetes, due to direct extension of the inflammation from the gall bladder to the pancreas. In diabetic persons over 25 years of age, gall stones are about 50 per cent more common than in non-diabetic persons.[13]. This may mean that gall stones and diabetes are both the result of disordered digestion of fat. We have, however, no definite proof of this at the present time. There is no way known to eradicate gall stones except by surgical removal of them and this removal may possibly prevent diabetes.

As far as we know at present, diabetes is not caused by any acute infection, but we do know that sometimes a very fulminative type of diabetes may develop after various forms of acute infectious disease[14]. This may be due to the fact that persons convalescing from such disease have huge appetites and are overfed, and in consequence put too heavy a load on the pancreas at a time when it is weak and needs nurturing rather than overloading. This would be especially true in a person in whom there was an hereditary tendency to diabetes. An Italian physician[15] reports that about half of his diabetic patients had chronic infections of some sort, which may have damaged the pancreas, thereby causing diabetes. For these reasons, it is wise to watch the urine closely during infectious disease and for several weeks following convalescence.

It is not possible to say that pregnancy is a cause of diabetes, but it is known that sugar very frequently appears in the urine of pregnant women. Whenever this occurs, that woman should be considered a potential diabetic, especially if diabetes is known to be present in her family. She should never allow herself to become fat and should have the urine tested twice each year.

A strenuous life, especially one in which nervous tension is continuously associated with heavy responsibilities not infrequently precedes the development of diabetes. Many diabetic children have been unusually brilliant in their school work, an accomplishment which usually involves great nervous tension. Careful supervision and adequate relaxation may prevent the development of diabetes in this group of young persons. In other cases, the disease has followed *injuries* which involved the pancreas. Sometimes *arteriosclerosis*, i.e., hardening of the arteries, seems to be the only antecedent cause of diabetes. If any of these conditions occur in a person who is fat and happens also to have a family tendency to diabetes, the likelihood of his developing the disease is very much increased. Thus, we see that several factors may be involved in the cause of any one case of diabetes, and prevention depends upon the control of these several factors.

The prevention of diabetes is an individual responsibility and will be achieved only at the expense of personal effort and inconvenience. The doctor cannot accomplish it for you, although he can help by thoughtful care during pregnancy and acute infections, and by intelligent treatment of disease of the gall bladder. The handicap of an hereditary tendency toward diabetes may be offset by the prevention of obesity. In conclusion, I cannot overemphasize the fundamental requirements in the prevention of diabetes, which are the will power to keep your weight at the average level or

below, and a physical examination, including urinalysis, at least once each year.

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COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1932 AND SEVEN YEAR AVERAGE

MONTH ENDING APRIL 29, 1933

Diseases	1933 Week ending April 8	1933 Week ending April 15	1933 Week ending April 22	1933 Week ending April 29	Average cases reported for week corresponding to April 29 for past seven years	1932 Week ending April 9	1932 Week ending April 16	1932 Week ending April 23	1932 Week ending April 30
Actinomycosis	—	—	—	—	—	—	—	1	—
Cerebrospinal Men.	—	1	—	—	1	2	1	—	—
Chicken Pox	159	136	190	158	72	117	87	78	106
Conjunctivitis Inf.	—	—	1	—	3	—	—	9	3
Diphtheria	4	5	5	7	16	6	8	8	5
Encephalitis Epid.	—	—	—	1	—	1	—	—	—
German Measles	9	4	7	6	31	4	5	5	7
Influenza	19	7	10	5	8	19	17	8	5
Measles	275	242	265	273	311	112	156	160	158
Mumps	117	88	86	84	74	92	67	80	66
Paratyphoid Fever	—	—	2	—	—	—	—	1	5
Pneumonia (Broncho)	19	22	22	24	32	49	27	26	38
Pneumonia (Lobar)	40	25	32	20	43	59	39	30	36
Poliomyelitis	—	—	—	—	—	1	—	—	—
Scarlet Fever	167	140	118	134	80	85	93	119	130
Septic Sore Throat	2	—	5	1	3	2	—	2	1
Smallpox	—	—	2	9	—	—	—	—	—
Tetanus	—	—	—	1	—	—	—	—	—
Trichinosis	—	1	—	—	—	—	—	—	—
Tuberculosis (Pul.)	34	15	37	30	37	23	22	40	38
Tuberculosis (O. F.)	1	2	2	3	4	1	5	2	1
Typhoid Fever	—	—	1	1	1	1	3	—	—
Undulant Fever	1	—	—	—	—	1	—	—	—
Whooping Cough	112	88	81	112	58	152	129	126	96
Gonorrhea	23	16	33	23	23	21	16	50	46
Syphilis	43	23	32	62	39	33	67	61	93

Remarks: No cases of Asiatic cholera, glanders, plague, or yellow fever during the past seven years.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending April 22, indicate a mortality rate of 11.0 as against a rate of 12.0 for the corresponding week of last year. The highest rate (18.0) appears for Washington, D. C., and the lowest (5.8) for Somerville, Mass. The highest infant mortality rate (10.5) appears for Fall River, Mass. (due mainly to hospitalization facilities), and the lowest for Kansas City, Kans., Miami, Fla., Springfield, Mass., Utica, N. Y., Wilmington, Del., and Yonkers,

N. Y., which reported no infant mortality.

The annual rate for 85 cities is 12.1 for the sixteen weeks of 1933, as against a rate of 12.6 for the corresponding period of the previous year.—*Bureau of the Census.*

HOSPITALS AND THE DEPRESSION

Those hospitals which are dependent on more or less constant donations are seriously handicapped at the present time. A considerable number have suspended.

An unfortunate illustration of these conditions is that of the New York Nursery and Child's Hospital at Sixty-First Street and Amsterdam Avenue, New York City, which is the only maternity hospital on the west side between Fifth Avenue and the Hudson River for one hundred blocks south of One Hundred and Ninth Street.

This is the oldest children's hospital in America and the oldest maternity hospital in point of service in New York City. The only possibility of continuing is an emergency fund of one hundred thousand dollars.

The closure of this hospital is regarded as an utter calamity by the Commissioner of Health.

This hospital is a health center of "child saving" for 400,000 people.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR APRIL, 1933

During the month of April the incidence of communicable diseases was at an extremely low level. Of special note were the complete absence of infantile paralysis and the low level for meningitis and diphtheria. In the case of the latter, the incidence was lower than had ever been previously reported for the month of April. During the middle of the month there were slight evidences of a recrudescence, but this was apparently short lived, the month ending with the disease at a record low figure.

Lower figures than ever previously recorded were noted for epidemic cerebrospinal meningitis, lobar pneumonia and pulmonary tuberculosis.

The decline in incidence of scarlet fever which has been noted since the beginning of the year was continued, indicating that the wave of this disease which has been going through the Metropolitan Area is distinctly on the decline. In certain parts of the state, notably the southern corner, there is, however, evidence of a continued increase in scarlet fever.

Rabies in dogs reached a surprisingly low figure for all sections of the state except Boston, where during the middle of the month there has been a sharp increase. It is too early to determine whether this will be continued.

RARE DISEASES

Actinomycosis was reported from North Reading, 1.

Dysentery was reported from Boston, 1; Danvers, 3 (State Hospital); Quincy, 1; Total, 5.

Epidemic Cerebrospinal Meningitis was reported from Chicopee, 1; Hingham, 1; Lowell, 1; Malden, 1; Peabody, 1; Salem, 1; Total, 6.

Paratyphoid Fever was reported from Brockton, 1.

Septic Sore Throat was reported from Belmont, 1; Boston, 2; Cambridge, 3; Duxbury, 1; Easton, 1; Lowell, 2; Melrose, 1; Newton, 1; Salem, 2; Walpole, 1; Total, 15.

Trachoma was reported from Boston, 3; Canton, 1; Wakefield, 1; Total, 5.

Trichinosis was reported from Northampton, 1.

Undulant Fever was reported from Holbrook, 1; Orange, 1; Total, 2.

MONTHLY REPORT FOR APRIL, 1933

Disease	Apr. 1933	Apr. 1932	Average*
Anterior Poliomyelitis	0	5	4
Chicken Pox	1209	997	855
Diphtheria	85	130	258
Dog Bite	590	514	483
Epidemic Cerebrospinal Meningitis	6	12	14
German Measles	92	100	411
Gonorrhea	388	474	422
Lobar Pneumonia	339	626	561
Measles	1974	3108	3880
Mumps	786	1385	881
Paratyphoid Fever	1	3	
Scarlet Fever	1648	2235	1470
Syphilis	339	369	259
Tuberculosis, Pulmonary	318	406	416
Tuberculosis, Other Forms	42	61	61
Typhoid Fever	12	7	13
Whooping Cough	840	898	875

*Average number of cases for April during the preceding five years.

CORRESPONDENCE

PATRONAGE OF SCHOOLS OF PUBLIC HEALTH

Harvard University
School of Public Health
55 Van Dyke Street,
Boston, Massachusetts,
May 12, 1933.

Editor,
The *New England Journal of Medicine,*

In the issue of April 13, 1933 of The *New England Journal of Medicine* there appears a letter by Dr. Victor Safford, Deputy Health Commissioner of the City of Boston. In the course of his letter Dr. Safford makes the following statement:

"The patronage of these schools (of Public Health) has been disappointing, both in regard to the number and the type of persons that have taken advantage of the educational opportunities thus offered. To a noteworthy extent the students have been foreigners whose tuition has been paid by the Rockefeller Foundation. Of the 32 gradu-

ates of the Johns Hopkins School of Public Health in 1931 who received the degree of Doctor of Public Health, seventeen were aliens. . . . Virtually, the same condition obtains with respect to the Public Health Schools located in Massachusetts."

As a member of the faculty of the Harvard School of Public Health, I cannot permit these statements to go unchallenged. The facts are as follows:

Since 1911 a total of 707 degrees in Public Health have been conferred by the Schools of Public Health connected with Johns Hopkins, Toronto and Harvard Universities. Most of these degrees were given after 1919. Three hundred and eighty-six degrees were conferred upon citizens of the United States, 84 on Canadians and 237 on students from foreign countries. If we consider Canadians as Americans, 33.5% of the degrees were granted to foreign students.

We are delighted to have these foreign students. They are carefully selected by their own governments, and on completion of their training, return to positions of responsibility in their own countries. With few exceptions, they have been a highly intelligent group of men.

In 1931, the year selected by Dr. Safford, Johns Hopkins granted the following Public Health degrees:

Dr.P.H.	15
Dr.Sc.	19
Certificate in P.H.	32
Master in P.H.	3
Total	69

Of this group, 57 were Americans, 5 Canadians and 27 foreigners.

My own class in Public Health Administration in the Harvard School of Public Health during the current academic year consisted of the following students: Americans, 19; Chinese, 2; British West Indies, 1. All were graduates of Class A Medical Schools, and were of a high average of intelligence and ability.

It is quite unfair to the Harvard School of Public Health and unjust to its students to state that its "patronage has been disappointing, both in regard to number and type of student". On the contrary, the school has had a steady, consistent growth, with gradual, but striking improvement in the quality of its students.

The school does not desire a large number of students. Rather it has seemed advisable to develop slowly, train the students well and limit the applicants to properly qualified men. The increasing interest and confidence in the school on the part of federal, state and municipal health authorities have been sources of the greatest satisfaction to the faculty.

Very truly yours,

WILSON G. SMILLIE, M.D.,
Professor of Public Health Administration, Harvard School of Public Health.

THE REPORTING OF INFLUENZA

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

May 10, 1933.

Editor,
The *New England Journal of Medicine,*

Since the reporting of influenza has served no useful purpose and in some instances has been a positive detriment, the Public Health Council today voted to withdraw this disease from the list of those that are reportable.

Because of the difficulties in an individual case in distinguishing between a severe cold, grippe and influenza, the determination of whether or not the particular condition should be reported is generally dependent upon the state of apprehension in regard to the disease at a given moment, particularly as manifested through the newspapers. One day we may have no influenza reported but if the newspapers headline on that day that the disease is threatening us many hundred cases may be reported the next day so that in general this reporting is an index of apprehension rather than of disease.

It is of course important in the institution of control procedures that the incidence of communicable disease be known currently as accurately as possible. During the influenza season this has been obtained through weekly reports as to the total incidence of respiratory disease in representative industries and teaching institutions over the State, from some one hundred physicians who have very kindly coöperated in reporting current experience in their practice, and from the case load of representative visiting nursing associations. In addition, the severity of the infection is currently checked through weekly reports by local boards of health of pneumonia deaths. From all these data we get a current clear picture of incidence and severity which is dependable and can be compared with previous years and on which necessary administrative procedures may be based.

Yours truly,

GEORGE H. BIGELOW, M.D.,
Commissioner of Public Health.

LETTERS RELATING TO THE OWNERSHIP OF THE *BOSTON MEDICAL AND SURGICAL JOURNAL*

The subjoined letters from former owners of the *Boston Medical and Surgical Journal* to Dr. Joel E. Goldthwait one of the group that purchased the *Journal* before these were written will be of interest to all who have followed the history of the *Boston Medical and Surgical Journal* up to its transfer to the Massachusetts Medical Society in 1921 with the later change of title to The *New England Journal of Medicine* in 1928. These letters are published through the courtesy of Dr. Goldthwait.

Older practitioners knew Dr. Shattuck, Dr. Rich-

ardson and Dr. Warren with appreciation of the public spirit which led to assuming the burden of maintaining the *Journal*.

For several years before taking over the ownership, the Massachusetts Medical Society subscribed to the *Boston Medical and Surgical Journal* for each member.

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225 Commonwealth Avenue
Boston

April 16/14.

Dear Doctor:

Many thanks for your very kind letter. I can only say that we owners could not have asked for better successors than you three gentlemen.

Wishing you good luck.

Sincerely,
W. L. RICHARDSON.

---

58 Beacon Street

April 17, 1914.

Dear Dr. Goldthwait:

I wish to acknowledge with many thanks your kind and sympathetic note, about the *Boston Medical and Surgical Journal*. I have felt, so far as I was concerned, that it was a heritage that I must do my share in passing along to others. I feel it has been a good thing that the profession of Boston has had some kind of an organ all of these years to represent it in medicine. Now that a period of unusual activity is to come, I should be very sorry to see the old *Journal* make way for some newly-fledged successor.

The weight of years, together with the infusion of new blood, such as you and your colleagues represent, will I feel sure, make it a valuable exponent of New England medicine and, perhaps, even of national medical science and practice.

With best wishes for the future, and much satisfaction in the feeling that it is in such good hands, I am,

Sincerely yours,
J. COLLINS WARREN.

Dr. Joel E. Goldthwait.

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135 Marlborough Street,

Boston, April 17, 1914.

Dear Goldthwait:

Thank you for your letter of the 15th. That you should think of and take the trouble of writing it is gratifying.

We were all of us glad to take a hand in the enterprise which seemed to be of benefit to the profession, and were amused, rather than distressed, that we should be supposed to be deriving great dividends from the *Journal*. It is comforting to be assured that there are others who are ready to take up the burden, and I have no doubt that the *Journal* will continue to be worthy of support under its change of ownership.

I think it should be known that my brother really did more than all the rest of us put together. He never received any compensation at all adequate to the amount and character of the work he put into the *Journal*, and for the last two, or I think three, years of his editorship took no compensation at all. This fact he would be the last person to mention; but it should be understood.

With best wishes,

Yours sincerely,
F. C. SHATTUCK.

Dr. Joel E. Goldthwait,
372 Marlboro Street,
Boston.

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225 Commonwealth Avenue
Boston

October 18/16.

Dear Doctor:

Many thanks for the check so generously sent. It is certainly a case of bread cast upon the waters.

I can congratulate the owners on the great improvement they have made in the *Journal*.

Sincerely,
W. L. RICHARDSON.

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135 Marlborough Street,
Boston, October 19, 1916.

Dear Goldthwait:

Your check comes as a pleasant surprise. I never expected to get anything back. I can use the money in my business, and am glad to be presented with such strong evidence of the success of the *Journal*.

Yours very truly,
F. C. SHATTUCK.

Dr. Joel E. Goldthwait,
372 Marlboro Street,
Boston, Mass.

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58 Beacon Street

October 20, 1916.

Dear Dr. Goldthwait:

I beg to acknowledge with many thanks your check for two hundred dollars as part payment of the money advanced by me for work of the *Medical Journal*.

This is not only most acceptable but encourages me to hope that the *Journal* is entering upon an era of prosperity, which speaks well for the present management.

With best wishes for the future, I am,

Sincerely yours,
J. COLLINS WARREN.

Dr. Joel E. Goldthwait.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
April 29, 1933.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of March 31 the following have been accepted:

Cutter Laboratory
Polyanaerobic Antitoxin, Prophylactic (Tetanus-Gas Gangrene Antitoxin)
Polyanaerobic Antitoxin, Therapeutic (Gas Gangrene Antitoxin)

E. Fougera & Co.
Capsules Lipiodol—Lafay, 0.5 Gm.
Tablets Lipiodol—Lafay, 0.04 Gm.

Merck & Co., Inc.
Calcium Gluconate—Merck
Phenobarbital Sodium—Merck

G. D. Searle & Co.
Sodium Morrhuate 5% with Benzyl Alcohol

Yours very truly,
PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

RECENT DEATHS

FIPPHEN—CLARENCE WYMAN FIPPHEN, M.D., of Shrewsbury, Mass., died at his home May 7, 1933. Dr. Fipphen was born in Concord, N. H., in 1895, and received his early education in that city. He graduated from Dartmouth College in 1916 and the Harvard Medical School in 1919. He served an interneship in the Worcester City Hospital and practiced in Worcester subsequently. During the war he served in the medical corps. He joined the Massachusetts Medical Society in 1919. He was also a Fellow of the American Medical Association.

Dr. Fipphen is survived by his widow Mrs. Ethel (Dole) Fipphen, two sons and a brother, Dr. Earl E. Fipphen of Worcester.

Dr. Fipphen was active in civic affairs having served on the school committee and at one time on the local board of health. He was affiliated with the Masonic fraternity and the Ray Stone post, A. L.

BROWN—ARTHUR LINWOOD BROWN, M.D., of 69 Church Street, Winchester, Massachusetts, died May 15, 1933. He was born in Fall River, Massachusetts, in 1869, the son of Arthur Taylor Brown and Elizabeth (Blackway) Brown.

His early education was acquired in Fall River with later studies in a Boston preparatory school. He graduated in medicine from the Tufts College Medical School in 1908 and engaged in postgraduate work at Harvard and Johns Hopkins Medical Schools after intern service at the Boston City Hospital. Subsequently he held the position of resident physician at the Malden Hospital. He joined the Massachusetts Medical Society in 1910.

Dr. Brown had practiced in Winchester continuously from 1910 except for the period of war service with the rank of Captain from October 1918, serving first at the training camp at Fort Oglethorpe, Georgia, later in field service at Hoboken and thence to Virginia terminating military service at the time of his honorable discharge in 1919. He was a member of the Parkman Lodge and Winchester Royal Arch Chapter of Masons, the Elks, the American Legion, and the Boston Square and Compass Club.

As President of the American Electrotherapeutic Association, he represented that organization at the meeting in Liege, Belgium. He was the author of various articles on physiotherapy published in *American Medicine* and the *Journal of Physiotherapy.*

Dr. Brown is survived by his mother and a son.

WALKER — WALDO WEBSTER WALKER, M.D., of Newtonville died at the Massachusetts Memorial Hospitals, May 15, 1933, after an illness of three months. He was born in Clatonia, Nebraska, in 1885, and graduated in medicine from the State University of Iowa College of Homeopathic Medicine in 1913. He came to Boston in 1914 and developed an important practice in nose and throat surgery and was the Assistant Professor of Laryngology and Rhinology at the Boston University School of Medicine. His office was at 99 Bay State Road, Boston.

He joined the Massachusetts Medical Society in 1918 and served with the Medical Corps at Asheville, N. C., during the World War. He was a member of the Dalhousie Lodge of Masons, the Newton Club, and the Central Club. He is survived by his widow, Mary (Brock) Walker, and two children Lloyd and Jean Walker.

NOTICES

CORRECTION

The spelling of the name of Mr. Romney Spring, Assistant-Treasurer of the Massachusetts Tuberculosis League, was incorrect as it appeared on page 1023, issue of May 11. The spelling is correct as it appears above.

COURSES AT THE TOMARKIN FOUNDATION

Conferences for the fifth International Medical Post-Graduate Courses of the Tomarkin Foundation will be held in St. Moritz, Switzerland, August 13-27, 1933.

The subjects in these courses are Allergic Diseases, Diseases of the Blood, Climatology, Rheumatism and Arthritis and Diseases of Metabolism. For detailed information apply to Chr. Jilli at St. Moritz.

CHICAGO MEDICAL SOCIETY WILL PROVIDE INFORMATION FOR VISITING PHYSICIANS

During the summer of 1933, The Century of Progress Exposition is being held in Chicago.

The Chicago Medical Society has a booth in the Hall of Science Building in Group K. In this booth we will have information for visiting physicians and will be glad to assist any of the members from other states in every way possible.

The Woman's Auxiliary of the Chicago Medical Society will welcome the wives and daughters of physicians.

I trust we will have the pleasure of meeting a large number of physicians and of being some help to them.

Yours very truly,

COMMITTEE—CENTURY OF PROGRESS.
WILBUR POST,
JULIUS HESS,
HUGH N. MACKECHNIE,
Chairman.

RADIO HEALTH MESSAGES

MAY-JUNE, 1933

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:30 P.M.

May
23 Chronic Bronchitis
30 Neurasthenia

June
6 Safer Maternity
13 The Care of the Skin
20 A Heart to Heart Talk
27 Social Service in Public Health

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:45 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

THE IVTH INTERNATIONAL CONGRESS OF RADIOLOGY

will be held in Zurich, under the presidency of Professor H. R. Schinz, from July 24-31, 1934.

According to §3 of the "Rules and Regulations" the Congress may be attended by:

1) Members of radiological societies in all countries.

2) Persons introduced by such societies.

The 32 countries which were represented at the IIIrd International Congress of Radiology in Paris have been invited by the Organizing Committee to appoint delegates to the International Committee and to nominate a speaker to report on the organization of the cancer campaign in his country at the opening session.

At the general meeting the following subjects will be treated:

X-ray Diagnosis of Bone Tumors
Vasography
The Development of Pulmonary Tuberculosis as seen radiologically
Radiation Treatment of Uterus-Carcinoma
Radiation Treatment of Malignant Tumors of the Mouth and Pharynx
Radiation Genetics
Mitogenetic Radiation (Mitogenetische Strahlung)
Structure Analysis
Identical Physical Measurement of the Dose in X-ray and Radium Treatment
Hard Gamma-Rays, Cosmic Radiation, Earth-Radiation
Shortwave Therapy (Kurzwellentherapie).

In addition the sections for X-ray Diagnosis, Radiotherapy, Radiobiology, Radiophysics and Radiotechnic, Electrology and Heliotherapy will hold discussions and every member of the Congress is entitled to present a communication.

During the Congress an exhibition of apparatus, photographical accessories, chemical products and scientific books will be on view. (For details apply to A. Strelin, Kilchberg/Zurich.)

All radiological societies are requested to send as soon as possible a list of their members to the General Secretary, Dr. H. E. Walther, Gloriastrasse 14, Zurich.

THE 1933 GRADUATE FORTNIGHT OF THE NEW YORK ACADEMY OF MEDICINE

Metabolic Disorders will be the theme of the 1933 Graduate Fortnight of the New York Academy of Medicine. Two weeks of intensive study, from October 23 to November 3 inclusive, will be devoted to this important branch of medical science. The theoretical, physiologic and pathologic phases of Metabolism, as well as of certain of the associated endocrinologic problems will be treated in a series of round table discussions and clinical demonstrations. The latter will be given in fifteen of the leading hospitals of New York City.

Among the speakers who will participate in the Graduate Fortnight are included Drs. Eugene F. DuBois, Harold E. Himwich, Walter W. Palmer, Frank H. Lahey, Donald Dexter Van Slyke, Joseph C. Aub, Ashley Weech, Dana W. Atchley,

Erwin Brand, Emanuel Libman, Rollin T. Woodyatt, Priscilla White, Nellis B. Foster, Herman O. Mosenthal, William S. Ladd, Henry W. Geyelin, Albert A. Epstein, John P. Peters, Henry C. Sherman, Samuel W. Clausen, Alfred F. Hess, Wilder G. Penfield, Oscar M. Schloss, Henry L. Jaffe and Charles F. Bodecker.

An exhibit will be shown in connection with the Fortnight, material having been collected from many institutions in Metropolitan New York. The various aspects of metabolic disorders will be covered in this exhibition including the history of metabolism; dietary constituents and their derivatives; drug and other therapeutic measures; general and special pathological metabolism; and laboratory methods and procedures.

The subjects will be illustrated by means of charts, graphs, photographs, microphotographs, transparencies, x-rays, gross and pathologic specimens.

The profession of the country is invited to participate in the Graduate Fortnight.

A complete program and registration blank may be secured by addressing Dr. Frederick P. Reynolds, The New York Academy of Medicine, 2 East 103rd Street, New York City.

REPORTS AND NOTICES OF MEETINGS

SUFFOLK DISTRICT MEDICAL SOCIETY

ANNUAL MEETING

The annual meeting of the Suffolk District Medical Society was held in the John Ware Hall of the Boston Medical Library on the evening of April 26 at 8:30 P.M. Dr. William H. Robey presided. The reports of the secretary, treasurer and auditing committee were read and a letter from Dr. Walter Bowers asking the Society for its support of a resolution dealing with the discretionary powers of the Massachusetts Medical Examining Board. The resolution was read and sent to a committee. The annual election of officers was held and the following were elected:

President: William H. Robey.

Vice-President: James H. Means.

Secretary: George P. Reynolds.

Treasurer: John Rock.

Commissioner of Trials: Channing Frothingham. Councillor for the Nominating Committee: Lincoln Davis. Alternate: Reginald Fitz.

Committee on Supervision: Stanley Cobb, Thomas H. Lanman.

Committee on Social Meetings: DeLos J. Bristol, Jr., Lester A. Whitaker, A. McK. Fraser.

Auditing Committee: Robert N. Nye, Francis C. Newton, Charles C. Lund.

Censors: Alonzo K. Paine, Horace Binney, George L. Tobey, Jr., Gerald Blake, William B. Breed.

Councillors: A. W. Allen, A. E. Austin, J. W. Bartol,* G. H. Bigelow, H. Binney, G. Blake, W. B. Breed, W. J. Brickley, J. E. Briggs, C. S. Butler, D. Cheever, R. C. Cochrane, F. H. Colby, F. J. Cotton, A. H. Crosbie, W. P. Cross, L. Davis, G. P. Denny, R. L. DeNormandie, W. H. Ensworth, R. Fitz, C. Frothingham, G. L. Gately, H. W. Goodall, R. B. Greenough,* J. Homans, H. T. Hutchins, E. P. Joslin, F. H. Lahey, R. I. Lee, G. A. Leland, T. J. O'Brien, A. K. Paine, F. W. Palfrey, W. S. Parker, W. F. Regan, G. P. Reynolds, W. H. Robey, G. C. Shattuck, W. R. Sisson, Louisa Paine Tingley, G. L. Tobey, Jr., J. R. Torbert, H. P. Towle, F. A. Washburn, C. Wesselhoeft.

Dr. Channing Frothingham read a paper on the "Organization of the Medical Profession for Rendering the Best Medical Service." The speaker stated that the Committee on the Costs of Medical Care had shown quite clearly that the amount of money spent by the people of this country for medical care is not unreasonable, but that it is unevenly distributed and a good deal of it poorly spent. How the redistribution of cost is to take place was not taken up, but the speaker centered his attention on the medical care which is the best for the people. He stated that this is the primary consideration and once this is determined the people should be educated to seek this for themselves.

To whom should the patient go when he wants medical advice? In outlining the answer to this query Dr. Frothingham showed the need for a general medical adviser who knows the environment of the patient as well as the organization of the medical profession. Such a person should be well grounded in his knowledge of medicine, should keep abreast of the new advances in his profession, and should diagnose and treat such conditions as are within his ability to handle well. The other cases should be guided into the proper channels for their care.

In enlarging upon this main theme of the general medical adviser, Dr. Frothingham considered the organization of the hospital out-patient departments and hospitals for treating special diseases. Instead of being seen in the medical or surgical division upon first entering the out-patient department, the patient should be admitted to a general diagnostic clinic, from whence he can be sent to whatever service can best treat his condition. Hospitals for treating special diseases tend to accentuate one particular phase of the patient and fail to depict the whole medical picture.

Dr. Richard M. Smith opened the discussion of this paper. He reviewed the gradual changes that have taken place in the medical profession during the past few years. Slowly the physician has segregated his office from his home; then a group has come together and used the same waiting room and secretarial staff; and finally there has grown up a division of labor within such a group. The tendency to use the hospital more and more has led to a tremendous increase in the hospital beds of this country, and Dr. Smith questions whether

*Councillor for life by virtue of being a past President of the State Medical Society.

this free use of the hospital is necessary. But with such a tendency there also evolves the specialized hospital.

The speaker stated that we cannot simply regret that these changes have taken place. We can be complacent toward them, but rather we should study the problem with an attempt to ascertain which trends are in the right direction and try to select these and develop them further. The practice of medicine is a personal relationship, and any organization that destroys such a relationship is undesirable. Keeping this in mind we should attempt to further any measure that makes for a better quality of medical service.

Dr. Frank H. Lahey mentioned the necessity of advancing slowly in any changes in the organization of the medical profession especially in view of the unsettled conditions at the present time. Whatever measures are adopted should be carefully scrutinized for possibilities of disrupting the already smooth running machinery of medical care. In certain clinics of the country visited by the speaker the undesirable feature of solicitation for patients was seen. In one city various clinics have begun price cutting. Regardless of what method of group practice is advocated, it should be free of any element of solicitation. Finally Dr. Lahey stated that he was heartily in favor of Dr. Frothingham's general medical adviser, but that he considered such a person quite difficult to find.

Dr. Hans Zinsser stated that in his opinion the most important problem to be settled in the reorganization of medical care is how it should be paid for. He believes that the changes discussed by Dr. Frothingham are taking place at present and that we must now decide how they should be paid for. We know that a certain percentage of the population cannot pay for medical care and that a certain number of people must be hospitalized. The medical profession should not care for these people without pay for their services. No other profession works so much for nothing.

The amount of money necessary to pay for medical services and hospital care rendered to the indigent could be easily computed and paid for out of taxes. Measures should also be taken to help out that great middle class which can well afford to pay the medical adviser, but cannot afford the fees of the specialist.

In organizing any scheme for medical practice it must be centered around the hospital. All doctors should be included and many hospitals brought within the group so that the choice of the patient may not be jeopardized. Unless the medical profession takes up some system of reform in the near future, it is possible that the economic interests of the country may take it up to great disadvantage to the profession at large.

Dr. Frederic A. Washburn stated that a definite proposal in relation to the periodic prepayment of hospital care was in preparation at the present time and would probably be proposed in the near future. He hoped that the medical profession would receive it with open mind.

A motion was then passed for the appointing of a committee to study any such proposal—including also prepayment for medical care during such hospitalization. This committee is to report and make recommendations at the next meeting, October, 1933.

WORCESTER DISTRICT MEDICAL SOCIETY

At the annual meeting of the Worcester District Medical Society held May 10, the following officers were elected:

President: Dr. Walter C. Seelye, Worcester.

Vice-President: Dr. Ernest B. Emerson, Rutland.

Orator: Dr. William F. Lynch, Worcester.

Treasurer: Dr. Edward P. Disbrow, Worcester.

Secretary: Dr. Erwin C. Miller, Worcester.

Councillors on Nominations: Dr. David Harrower, Worcester; Dr. Royal P. Watkins, Worcester—Alternate.

Committee on Funds: Dr. Michael F. Fallon, Worcester; Dr. Royal P. Watkins, Worcester; Dr. Leslie R. Bragg, Webster.

Commissioner on Trials: Dr. Walter P. Bowers, Clinton.

Councillors: Dr. James C. Austin, Spencer, term began 1930; *Dr. Walter P. Bowers, Clinton, term began 1902; Dr. Leslie R. Bragg, Webster, term began 1922; Dr. Frank H. Clapp, North Grafton, term began 1930; Dr. Philip H. Cook, Worcester, term began 1929; Dr. William J. Delahanty, Worcester, term began 1913; Dr. William E. Denning, Worcester, term began 1933; Dr. George A. Dix, Worcester, term began 1921; Dr. George E. Emery, Worcester, term began 1920; Dr. Michael F. Fallon, Worcester, term began 1916; Dr. Homer Gage, Worcester, term began 1906; Dr. James J. Goodwin, Clinton, term began 1921; Dr. David Harrower, Worcester, term began 1905; Dr. Ernest L. Hunt, Worcester, term began 1918; Dr. Edwin R. Leib, Worcester, term began 1932; Dr. Arthur W. Marsh, Worcester, term began 1922; Dr. Erwin C. Miller (Secretary), Worcester, term began 1929; Dr. Joseph W. O'Connor, Worcester, term began 1931; Dr. Walter C. Seelye (President), Worcester, term began 1930; Dr. Albert M. Shattuck, Worcester, term began 1931; Dr. Edward H. Trowbridge, Worcester, term began 1924; Dr. Frank H. Washburn, Holden, term began 1916; Dr. Royal P. Watkins, Worcester, term began 1927; *Dr. Samuel B. Woodward, Worcester, term began 1902.

Censors: Dr. George A. Dix, Supervising Censor, Worcester; Dr. Winifred M. Grant, Worcester; Dr. Gardner N. Cobb, Worcester; Dr. John W. O'Meara, Worcester; Dr. John E. Talbot, Worcester.

*Councillor for life by virtue of being a past President of the State Medical Society.

Nominating Committee: Dr. Harold V. Williams, Whitinsville; Dr. Charles A. Sparrow, Worcester; Dr. John Fallon, Worcester; Dr. Alvin R. Moses, Charlton; Dr. Raymond H. Goodale, Worcester.

Library Committee: Dr. Raymond W. Cutler, Worcester; Dr. Gilbert W. Haigh, Worcester; Dr. James C. McCann, Worcester.

Librarian: Dr. Albert C. Getchell, Worcester.

Auditing Committee: Dr. Harry B. Goodspeed, Worcester; Dr. Stewart M. Gibson, Southbridge; Dr. Frank B. Carr, Worcester.

THE ALPHA OMEGA ALPHA

The annual banquet of the Alpha Omega Alpha chapter of Harvard University was held on Thursday evening, April 20, in the Aesculapian Room of the Harvard Club. Mr. Robert Wilson was chairman. He called upon Dr. C. K. Drinker to introduce the speaker of the evening, Dr. Alfred N. Richards of Philadelphia, who spoke on "The Physiology of the Kidney."

The experiments described were those recently made by the speaker in collaboration with Dr. A. M. Walker and their colleagues which had their origin in discoveries that the frog's kidney can easily be arranged for microscopic study during life (1920) and that it is possible to withdraw fluid from a single structural unit by means of capillary pipettes. The possibility is thus offered of examining urine in different stages of its elaboration from blood.

Qualitative tests have shown that glomerular fluid taken from the capsules of Bowman in frogs, necturi (H. L. White) and snakes resembles a protein-free filtrate from blood plasma. Quantitative study has been delayed by the necessity of devising manipulative technique for handling the exceedingly small amounts of fluid obtainable (0.0001-0.001 cc.) without contamination or loss and of adapting existing methods of quantitative chemical analysis to these small amounts of material. This has been accomplished and chlorides, urea, dyes, uric acid, glucose, phosphates and creatinine have been determined in glomerular fluid and plasma taken from the animals mentioned. The results show clearly that the fluid separated from the blood as it flows through the glomerular capillaries has the composition of an ultrafiltrate from plasma.

The development of these methods makes possible the quantitative study of changes which the glomerular filtrate undergoes during its passage through the tubule. For this work thus far necturi have been used. It has already been found that the reabsorption of glucose takes places from the proximal convoluted tubule; preferential reabsorption of chloride does not. Concentration of urea, phosphates, uric acid and creatinine begins, but is by no means completed there. Acidification of the urine (frog) takes place in the distal tubule.

The speaker stated his belief that there is justification for believing that the results of study of glomerular fluid in these lower animals are applicable to glomerular fluid in mammals, but was obviously cautious in applying results of experiments with necturus tubules to those of other species.

AMERICAN PSYCHIATRIC ASSOCIATION

THE EIGHTY-NINTH ANNUAL MEETING

Hotel Statler, Boston, Mass.

May 29, 30, 31, June 1, 2, 1933

OFFICERS

James V. May, M.D., President, Boston, Mass.

George H. Kirby, M.D., Vice-President, New York, N. Y.

Morgan B. Hodskins, M.D., Hon. Vice-President, Palmer, Mass.

Clarence O. Cheney, M.D., Secretary-Treasurer, New York, N. Y.

COMMITTEE ON ARRANGEMENTS

L. Vernon Briggs, M.D., Chairman, Boston, Mass.

Winfred Overholser, M.D., Secretary, Room 109, State House, Boston, Mass.

HOTELS

The Committee on Arrangements recommends the use of the headquarters hotel, the Hotel Statler, which has 1,300 rooms with which it can accommodate all members of the Association. Members of the Association will make their own reservations directly with the hotel.

Hotel Rates:

The Hotel Statler, Arlington Street at Park Square—Single room with bath, $3.50-$8.00; double room with bath, $6.00-$10.00. Ample parking space for automobiles is available in connection with and close by the hotel.

Hotels adjacent to the headquarters hotel:

Touraine, Boylston at Tremont Street — Single room, $3.00, up; double room, $4.50, up.

Westminster, Copley Square—Single room with bath, $2.50, up; double room with bath, $4.00-$5.00.

The Copley-Plaza—Single room with bath, $4.00-$8.00; double room with bath, $7.00-$12.00.

RAILROAD RATES

Railroad Associations have agreed to allow one and one-half round trip railroad fare rates to those who attend the meetings, provided not less than 100 members purchase railroad tickets. To secure reduced rate, members of the Association, when purchasing tickets to Boston for themselves and members of their families, should state that they are attending the meeting of the American Psychiatric Association and should without fail, obtain a *Certificate* (not a receipt) for each ticket. Such certificates must be presented at the headquarters registration desk for counter-signature by the Secretary and validation by the railroad representative. Tickets for return by the same route may then be purchased at one-half fare rate. One-

way tickets to Boston, and *Not* round trip tickets, should be purchased at the starting point.

The Committee on Arrangements is making special provision for entertainment of members of the Association and their guests. Facilities for golfers will be made available. Visits to the Wayside Inn and to Quincy and Plymouth are planned for the visiting ladies, as well as the opportunity of visiting other places of historical interest. McLean Hospital and various nearby state hospitals will welcome visits from members and guests.

The American Association for the Study of the Feebleminded will hold its annual meeting at the Hotel Statler from Wednesday, May 31, to Saturday, June 3.

The American Psychoanalytic Association will hold its annual meeting at the Hotel Statler on Wednesday afternoon, May 31.

PRELIMINARY PROGRAM

MONDAY, MAY 29

Morning and afternoon sessions of the Section on Convulsive Disorders

TUESDAY MORNING, MAY 30

Reports.
President's Address.

TUESDAY AFTERNOON, MAY 30

Section I: Studies in Pathology.
Section II: Adequate Psychotherapy in Public Mental Hospitals.

WEDNESDAY MORNING, MAY 31

Joint Session with the American Psychoanalytic Association.

WEDNESDAY AFTERNOON, MAY 31

Section I: Joint Session with the American Association for the Study of the Feebleminded.
Section II: Interpretation of Symptom Syndromes.

WEDNESDAY EVENING, MAY 31

Annual Dinner

THURSDAY MORNING, JUNE 1

Section I: The Work of the Institute for Child Guidance; Review of five years' experience.
Section II: Miscellaneous Topics.

THURSDAY AFTERNOON, JUNE 1

Section I: Studies in Pathology.
Section II: Treatment of Special Conditions.

THURSDAY EVENING, JUNE 1

Round Table Meetings:

The following subjects can now be announced:

The Management of Problems of Decreased Support.
Mental Defectives in Public Schools.
Occupational Therapy.
Parole and Colony Measures.
Private Schools for the Mentally Defective.
Problems in Criminality.
Psychiatric Nursing.
Psychiatric Social Work.
Research in Mental Defect.
Researches on Emotion.
The Rôle Played by Psychoanalysis in Unravelling the Meaning of Psychotic Symptoms.
Types of Psychotherapy.
Veteran's Administration.

Other Round tables can be arranged on request.

FRIDAY MORNING, JUNE 2

Section I: Phases of Education.
Section II: Criminology.
Introduction of President-Elect.
Adjournment.

PRELIMINARY LIST OF READERS

Dr. Felix M. Adams, Dr. Bernard J. Alpers, (By Invitation), Dr. C. H. Anderson, Dr. J. Fremont Bateman, Dr. Lauretta Bender, (By Invitation), Dr. Malcolm A. Bliss, Dr. Earl D. Bond, Dr. Karl Murdock Bowman, Dr. Louis J. Bragman, Dr. George C. Brink, (By Invitation), Dr. Isra T. Broadwin, Dr. Walter Bromberg, Dr. Walter Bruetsch, Dr. Ethel Friedman Buchman, (By Invitation), Dr. F. A. Carmichael, Dr. John Chapman, (By Invitation), Dr. Thomas B. Christian, Dr. Stanley Cobb, Dr. Isador H. Coriat, Dr. William Dameshek, (By Invitation), Dr. George E. Daniels, Dr. George Van Ness Dearborn, Dr. Lucille Dooley, Dr. Henry Byron Elkind, Dr. Armando Ferraro, (By Invitation), Dr. Daniel H. Fuller, Dr. Nicholas Gotten, (By Invitation), Dr. Alfred Gordon, Dr. Ransom A. Greene, Dr. Hugh S. Gregory, Dr. Roscoe W. Hall, Dr. Milton A. Harrington, Dr. Ives Hendrick, Dr. Lewis B. Hill, Dr. Leland E. Hinsie, Dr. Richard H. Hutchings, Dr. George Franklin Inch, Dr. Eugen Kahn, (By Invitation), Dr. Jacob Kasanin, Dr. Siegfried E. Katz, Dr. John A. Larson, Dr. Samuel Leopold, Dr. David M. Levy, Dr. Erich Lindemann, Dr. A. Sandor Lorand, Dr. Lawson G. Lowrey, Dr. James Lincoln McCartney, Dr. William Malamud, Dr. H. Houston Merritt, (By Invitation), Dr. Joseph C. Michael, Dr. Joseph J. Michaels, (By Invitation), Miss Katharine Moore, (By Invitation), Dr. Merrill Moore, (By Invitation), Dr. Abraham Myerson, Dr. Ralph A. Noble, (By Invitation), Dr. Clarence A. Patten, Dr. Horatio M. Pollock, Dr. George A. Preston, (By Invitation), Dr. Curtis T. Prout, Dr. David Rothschild, Dr. Paul Schilder, Dr. Olive M. Searle, (By Invitation), Dr. Lowell S. Selling, Dr. Meyer Solomon, Dr. Walter B. Swift, (By Invitation), Dr. Lloyd J. Thompson, Dr. Simon Tulchin, (By Invitation), Dr. Willem van de Wall, (By Invitation), Dr. William A. White, Dr. John C. Whitehorn, Dr. Lawrence F. Woolley, Dr. Morris Yorshis, (By Invitation), Dr. Lloyd H. Ziegler, Dr. Gregory Zilboorg.

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

The Annual Meeting will be held at the Copley-Plaza, on Wednesday, May 24. Dinner at 6:30 P.M., $2.50 per plate.

Election of Officers.

Communications:

1. "Aid Obtainable from the Department of Rehabilitation (Commonwealth of Massachusetts) for Disabled Persons." Mr. Herbert A. Dallas, Supervisor of Rehabilitation, State Department of Education.
2. "Current Medical Affairs." Thomas J. O'Brien, M.D.
3. "Medical and Hospital Fees under Workmen's Compensation Act." Francis D. Donoghue, M.D.

WILLIAM EDWARD BROWNE, M.D., *Pres.*,
WILLIAM PEARCE COUES, M.D., *Sec'y.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

May 18—New England Hospital for Women and Children. See page 1024, issue of May 11.

May 19—The New England Roentgen Ray Society. See page 1025, issue of May 11.

May 19—New England Pediatric Society. See page 1025, issue of May 11.

May 22, 23, 25, 26—Special Lectureship Under the Auspices of the New England Heart Association. See page 1022, issue of May 11.

May 24—Massachusetts Society of Examining Physicians. See notice above.

May 29, 30, 31, June 1 and 2—American Psychiatric Association. See page 1074.

June 12—Golf for Members of the American Medical Association. See page 1022, issue of May 11.

June 12-13—The American Proctologic Society. See page 1025, issue of May 11.

June 12-16—Woman's Auxiliary to the American Medical Association. See page 815, issue of April 13.

June 13—The American Board of Obstetrics and Gynecology. See page 607, issue of March 16.

June 13—American Heart Association. See page 768, issue of April 6.

July 24-31—The IVth International Congress of Radiology. See page 1071.

June 28 to July 3—Third International Hospital Association. See page 811, issue of April 13.

August 10, 11, 12—American Delegation to the International Goiter Conference. See page 768, issue of April 6.

August 13-27—Courses at the Tomarkin Foundation. See page 1070.

October 9-12—American Public Health Association. See page 403, issue of February 16.

October 23 - November 3—The 1933 Graduate Fortnight of the New York Academy of Medicine. See page 1071.

April 16-20, 1934—The American College of Physicians. See notice on page 970, issue of May 4.

BOOK REVIEWS

Synopsis of Gynecology. Based on the Textbook *Diseases of Women.* By HARRY STURGEON CROSSEN, M.D., F.A.C.S., and ROBERT JAMES CROSSEN, M.D., St. Louis: The C. V. Mosby Company. 1932. Price $2.75.

This convenient pocket-manual, based on the senior author's larger and familiar textbook, "*Diseases of Women*," presents in compact, compendious, succinct, and systematic form the facts of gynecology. It should be of particular value, first to those students who specialize in gynecology, as a means of memorizing and tabulating their knowledge; second to those who do not so specialize, as a means of obtaining an adequate general knowledge of the subject without the expense or labor of studying the larger volume; third to practitioners, as a means of reviewing the outstanding features of a specialty with which they are bound to come into frequent contact. The book is well illustrated with 109 excellent figures, and with an engraved full-page frontispiece plate which deserves particular praise as a reconstructed, semi-diagrammatic, synthetic median sagittal section of the female lower abdomen and pelvis, from original drawing by Dr. R. Walter Mills, correcting certain conventional and traditional anatomic concepts. The authors are to be congratulated and thanked by the profession for their admirable work.

Diseases of the Skin. By the late ROBERT W. MACKENNA. Third edition revised and enlarged by Robert M. B. MacKenna. William Wood and Company, Baltimore, 1933. 506 pages. Price $8.00.

MacKenna's third edition, the third in ten years, has been revised and published by the son of the former author. The book has thirty-six more pages, and nine more colored plates. The whole book has been revised, the subject of seborrheic dermatitis has been almost rewritten, and there are numerous references which were not a part of the previous editions. There are numerous tables on differential diagnosis, the colored plates are particularly good, and the whole field is covered well in brief and concise manner. It is written particularly for the student and the general practitioner. It is valuable to American students in conveying the attitude of English dermatologists and their treatment, to our physicians.

The Interpretation of Dreams. By PROF. SIGMUND FREUD. New York: The Macmillan Company, 1933. Price $5.00.

This well-known book has appeared in two English editions previously. This, the latest edition, is translated from the eighth German and is said to be thoroughly revised. The bibliography, however, is not carried beyond 1913, the date of the second English edition. The book is too well known to need comment, although attention should be drawn to the extraordinary foreword by the author appended to this edition: "This book, with the new contribution to psychology which surprised the world when it was published (1900), remains essentially unaltered. It contains, even according to my present-day judgment, the most valuable of all the discoveries it has been my good fortune to make. Insight such as this falls to one's lot but once in a lifetime."